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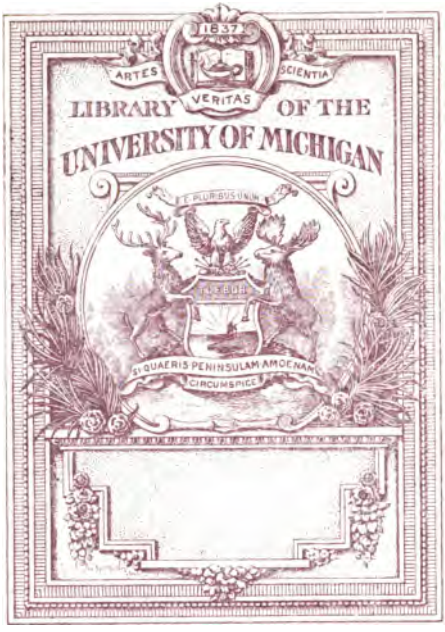

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TRANSACTIONS

OF THE

NINTH ANNUAL MEETING

OF THE

American Gastro-Enterological Association

1906

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Reprinted from the New York Medical Journal for March 9, 1907.

THE MUTUAL OBLIGATIONS OF SURGEONS AND INTERNISTS IN THE DEVELOPMENT OF GASTROENTEROLOGY.*

By Henry Wald Bettmann, M. D.,
Cincinnati.

The medical treatment of disorders of digestion has made enormous strides during the past twenty-five years. The chief factor in this progress has been the stomach tube. By means of the tube, diagnosis has been improved and thereby the adaptation of the treatment to the existing pathological conditions has been made possible. Other important factors have been the advance in our physiological conceptions of digestion and the facts revealed by abdominal surgery.

Unfortunately this knowledge and power have not become the common property of the profession. The general practitioners have not kept pace with the advances in this field of work. The surgeons, for the most part, have remained in ignorance of what can be accomplished by the proper treatment of digestive disorders. The cause of this state of affairs is not hard to find. The medical colleges up to within the past few years have not established special chairs in this department of practice. The advances in gastroenterology have appeared to the older practitioners to be of a too technical character for them to master. The internists who have been especially interested in this field have written comparatively little, internists in contrast to surgeons being possessed of what Sahli aptly called a

* Presidential Address at the ninth annual meeting of the American Gastroenterological Association, held at Boston, June 4, 1906.

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philosophical reserve—"Ein philosophisches Publications-Abstinenz." The Zeitgeist, too, pays rather indifferent attention to the plodding student of symptoms and signs, and encourages the bolder and more sensational work of the operator. This is well shown in the medical journals which minutely abstract the surgical papers and often slight, in stepmotherly fashion, the technical articles of the internist. It thus happens that the general practitioner when face to face with a difficult case has his train of thought diverted from the laborious channel of technical study into the more obvious path of surgical intervention. In this course he is encouraged by the surgeons who on the one hand have remained somewhat ignorant of the resources of internal medicine, and on the other are possessed of what an eminent surgeon has called "a magnificent optimism" regarding the results of surgical treatment. Thus the patient goes from bad or rather inadequate medical treatment, not to good or adequate medical treatment, but to surgery.

It is obvious that the reform of this unfortunate state lies in referring the obstinate cases not to the surgeon, but to the internists who by training are qualified to grapple with them. The cry should be "not from bad treatment to the surgeon, but from bad treatment to good treatment." The time will come when the general practitioner will be sufficiently educated to treat the severer gastric disorders successfully. Good early treatment will cure most cases. This statement cannot be too strenuously insisted upon. By good early treatment most of the surgical complications will be prevented. The surgeon must be taught to ask not *how long* the patient has been treated, but *how well.* The indications for operating should be based not on the obstinate nature of the case under bad treatment, but its resistance to appropriate treatment.

It is the daily experience of specialists to see

cases which have remained stubborn for months or years yield quickly and smoothly when an appropriate treatment is instituted. The advice to operate should come from the qualified internist and from no one else.

The internists will become qualified only when they are well taught. The greatest present needs in the department of gastroenterology are: 1. Systematic and specialized undergraduate instruction. 2. Wider opportunities for postgraduate study. 3. The education of the surgeons to a realization of the actual resources of internal treatment.

It is certainly a reproach to the profession when a celebrated surgeon[1] repeatedly states that dyspeptic patients will soon have to seek surgical treatment at first hand instead of medical treatment. That the surgeons need enlightenment may be inferred from a recent utterance of a surgeon[2] "that he was not acquainted with any momentous changes which have occurred in the treatment of digestive disorders throughout many generations." When the same surgeon declares from an experience of thirty operated cases "that persistent gastric indigestion is never functional in character merely," may we not somewhat wonder at the scientific acumen which permits of such broad generalization?

During the past few years many statistics have been gathered for the purpose of studying the frequency of gastric ulcer and the ultimate outcome of its medical treatment. Much serious study in this direction is urgently called for. American surgeons have exposed themselves to severe criticism for their reckless application of foreign statistics to American conditions. We need not go back so very far to find a famous surgeon writing that 4 to 5 per cent. of all people have gastric ulcer, and that 20 per cent. of these die as a result thereof. The incidence of gastric ulcer should be studied in every sec-

[1] Mayo. [2] Deaver.

tion of the country, in the dead room, in the clinic, and in the operating room. The autopsy records are not of themselves conclusive, for, as Brooks has pointed out, many ulcers of slight extent occur and heal without producing symptoms marked enough to induce the patient to consult a physician. Thus the pathological records will outnumber the clinical ones. The best source of correct statistics will be the medical wards of carefully conducted hospitals.

Regarding the final outcome of medical treatment, the last word has not yet been said. Results in private practice will differ tremendously from the results in hospital practice. Comparatively few private patients properly treated die of gastric ulcer. The earlier the treatment is begun, the less will be the mortality and the more satisfactory the permanent outcome. The average mortality will be less than 4 per cent. Hospital patients come under treatment in neglected condition, and proper after treatment is not possible. The ultimate prognosis in hospital cases under medical care is therefore doubtful, and a large proportion of those who leave the hospital in good condition will suffer from relapses and some from fatal complications.

Sufficient time has not yet elapsed for us to know the final outcome of operations for gastric ulcer in hospital patients. Reports are few, the total number of cases is small, and we have not as yet any detailed statistics by surgeons as to the ultimate fate of their hospital patients. It is possible that patients who are to be exposed after leaving the hospital to the physical hardships associated with poverty will do better under surgical than under medical treatment, although a final conclusion at the present time is not warranted by any available data. The careful nonpartisan collection of statistics through many years is required to make a definitive answer possible.

Internal medicine will undoubtedly be a great gainer through the increase of abdominal operations. The "autopsy in vivo" will clear up some dark chapters and may correct some faulty conceptions. Certain questions of diagnosis can be solved by the surgeons alone because all elements of doubt can be eliminated after the abdomen has been opened. Up to the present time the surgical literature has not been of material service in that regard.

I think we may fairly put it down as a grievance against the surgeons that they have given us few analytic studies of their operated cases, that they have overstated the difficulties and limitations of the medical diagnosis, and that they have indulged in careless generalizations. The typical cases are already well understood by the internists. Only the surgeons can give us definite statistics about the atypical cases, and aid the cause of diagnosis by an analytic study of all the symptoms and signs. Certainly the surgeons themselves cannot long remain satisfied to have "the majority of operations primarily exploratory" in nature based on chronicity or obstinacy of the symptoms.

Much confusion could be avoided if surgeons were to discuss the medical aspects of gastric disorders with more reserve. We are told by Mumford that it has been demonstrated that hyperchlorhydria is not the cause of heartburn. Deaver says that he has never seen cases of gastroptosis except in connection with dilatation of the stomach, or in that "rare" condition called splanchnoptosis. McGraw says that in cases of pyloric obstruction bile is always absent from the ejecta. Many absurd errors are found in the recent surgical literature on the stomach. These have been fully referred to in a previous article. A suggestion is made to surgeons to acquaint themselves thoroughly with the medical aspects of gastric disorders as a proper preparation for missionary work in surgery.

The darkest chapter in internal medicine is that on cancer of the stomach. The problem is one of early diagnosis so that a hopeless medical disease may become a promising surgical one. Late surgical interference accomplishes little or nothing. Palliative surgical measures are of doubtful utility except in a few cases.

The cases must be sent to the surgeon in time for the total extirpation of the diseased parts. The demand for early explorations in suspicious cases is wholly justified. But here again a reservation must be made. The suspicion indicating laparotomy must be in the mind of a qualified and experienced internist. If possible, surgery must be saved from the error of indiscriminate operating. Too many negative findings or futile operations will bring exploratory operations into disrepute. Surgeons should be frank enough to give us fuller statistics than is their wont. Cases of achylia gastrica and of atrophic gastritis will easily mislead the practitioner and the surgeon.

The most urgent need at the present time is to combat the furor operandi on the one hand and to make justifiable explorations popular on the other. Just how much we may expect from surgical interference in cases of cancer of the stomach lies with the future to decide.

LEVERONE BUILDING.

Reprinted from MEDICAL RECORD, September 1, 1906.

REMARKS ON BANTI'S DISEASE (SPLENOMEGALIA AND CIRRHOSIS OF THE LIVER).*

BY MAX EINHORN, M.D.,

NEW YORK.

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IN 1894 Banti[1] described four cases of splenomegalia, anemia, cirrhosis of the liver, and ascites, which ended fatally and came to an autopsy. From observations made on these and six other similar cases Banti described the clinical picture of the disease, which is at present universally known by his name. The essential part is a combination of splenomegalia, anemia, cirrhosis of the liver, and finally ascites. Although all these clinical manifestations were known, it was formerly customary to classify them according to the predominance of one or the other symptoms. It was Banti who first pointed out the relation of these various phenomena to each other.

According to Banti the disease has three stages: The preascitic or anemic, the intermediary, and the ascitic stage. Symptoms of the first stage are anemia and swelling of the spleen; increasing weakness, pallor of the mucous membranes, and dyspnea on

*Read before the American Gastroenterological Association on June 4, 1906, at Boston, Mass.

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exertion follow. Sometimes epistaxis occurs, or if there is severe anemia a transitory edema of the malleoli and an irregular intermittent fever result.

The blood shows diminution of the number of red blood corpuscles, a still more marked decrease in the amount of hemoglobin and poikilocytosis. The leucocytes are normal in every respect.

The anemia sometimes grows better, sometimes worse; it is amenable to rational treatment (arsenic, iron).

The swelling of the spleen develops slowly. When the patient consults a physician it has usually reached such dimensions that it represents a large tumor. It retains in general the rounded shape of the spleen, but is of a firmer consistency, and usually projects into the left abdominal cavity to a point somewhere between the navel and iliac crest. Banti thinks there is no doubt at all that the swelling of the spleen precedes the anemia. Liver and kidneys are normal. The first or preascitic stage lasts from one to four and a half years.

Now comes the second or transitional stage. At this time the urine is passed less frequently, and dyspeptic symptoms, like diarrhea, hemorrhoids, etc., arise. This stage is relatively short and lasts only a few months.

The third or ascitic stage begins with distention of the abdomen and ascites slowly develops. The fluid is never absorbed. Even after puncture the fluid will reaccumulate again very soon. At this time a decrease in size of the liver dullness can be demonstrated, the spleen remaining unchanged. The urine is diminished in quantity, concentrated, contains much urobilin, but no bile pigments, no albumin, and no sugar. The skin is of a somewhat darker hue and the conjunctiva somewhat icteric.

The signs of anemia slowly increase, and slight

rises of temperature in the evening occur. The patient becomes progressively weaker, and unless some other disease carries him off he dies from exhaustion.

The last stage is relatively short, lasting generally only a few months, at the highest seven to eight.

Pathologically a large amount of connective tissue proliferation was found in the spleen (fibroadenia) and in the liver (cirrhosis).

The well-known Italian investigator assumes that the spleen is the original seat of the disease, poisonous substances developing in it and entering the blood, in this way causing anemia and hyperplasia of the connective tissue in the liver. In some cases Banti has had the spleen extirpated with happy results.

Senator[2] later made the same subject the object of his investigation. He also classes the cases of splenic pseudoleukemia in the group of Banti's disease. Etiologically, Senator believes that autointoxications play a large rôle, because in many of these cases the splenic enlargement was preceded by diarrhea.

Regarding the symptoms Senator lays stress upon the hemorrhagic diathesis, which often occurs here, as evidenced by hemorrhages of the stomach and bowel, these being frequently the first sign to arouse the attention of the patient. According to Senator the ascites is sometimes absorbed. The blood is characterized by oligocythemia, oligochromemia (possibly leukopenia).

Curschmann[3] and Maixner[4] then pointed out the intimate connection between cirrhosis of the liver and profuse and sometimes fatal gastric and intestinal hemorrhages. In Curschmann's case the spleen was considerably enlarged, felt hard, was not sensitive to pressure, and projected beyond the border of

the ribs by about four fingers' width. The liver was somewhat small, but plainly cirrhotic.

Curschmann explains the hemorrhages by the fact that in cirrhosis of the liver collateral communications between the portal system and the system of the vena cava occur (thus causing varices in the stomach, esophagus, and pharynx).

Maixner has observed seven cases in which hemorrhages from the intestinal tract were the first symptoms to appear during the course of the disease, and also frequently to recur. In all cases the hemorrhage was very severe; in some even the direct cause of death.

In all these cases the liver is considerably enlarged and reaches nearly a hand's width below the right margin of the ribs. The spleen is also considerably enlarged and of hard consistency. Ascites was present only a few times. At autopsy there were no ulcers found in the stomach, only dilated veins in the cardiac part and in the esophagus. Maixner explains these symptoms as follows: "Early and frequent hemorrhages from the intestinal tract, large splenic tumor, light and slow development of ascites, and continuous diarrhea can be easily explained by the presence of a collateral circulation, accompanied by venous stasis. Since this circulation is one of the most perfect, we can see why the fatal course of liver cirrhosis can be so long protracted."

The resemblance of these cases of Maixner with those of Banti is evident. The gastric and intestinal hemorrhages have been counted as one of the symptoms of Banti's disease by Senator long before Curschmann and Maixner.

Von Aldor[6] lately described four analogous cases of "hemorrhagic cirrhosis of the liver" and discussed the question, whether these cases are not identical with Banti's disease.

Under this heading also falls an important article by William Osler, who, two years before Senator, Curschmann, and Maixner, pointed out the occurrence of severe gastric and intestinal hemorrhages in splenomegalia. Osler[6] calls his paper "Splenic Anemia," and emphasizes the fact that in these cases there was no cirrhosis of the liver. In perusing the histories, however, we often find that the liver is enlarged and projects several fingers' width beyond the free border of the ribs. The blood shows moderate diminution of the erythrocytes, absolute diminution of hemoglobin, and frequently leukopenia. Osler's cases resemble clinically those of Maixner and Aldor to such a degree that they will have to be placed under the same category.

Preble[7] about the same time describes four cases of hemorrhage from the stomach and bowels in cirrhosis of the liver, and tabulates fifty-six other cases. In 80 per cent. of these cases esophageal varices were found. In his own four cases splenic tumor, cirrhosis of the liver, and ascites were present. The cirrhosis of the liver may also take the form of the hypertrophic variety.

After having given some of the principal publications on Banti's disease and some of its varieties, I would like to relate a few of my own cases.

Not including the cases of pure splenomegalia (the splenic form of Hodgkin's disease) I saw in the last twelve years eighteen cases, which belong clinically to the category of Banti's disease. I have tabulated them and placed them in three groups:

1. Pure form: splenomegalia, anemia, cirrhosis of liver, ascites.

2. Hemorrhagic form, showing, in addition to the symptoms just enumerated, gastric and intestinal hemorrhages.

3. Splenomegalia, enlargement of the liver, ane-

mia, sometimes associated with grave gastric symptoms.

TABLE OF CASES OF BANTI'S DISEASE TREATED BY EINHORN DURING 1894–1906.

GROUP I.

No.	Name.	Age.	Date.	Principal Symptoms.	Course.
1	Carl B.....	15	1897	Marked enlargement of spleen and liver, anemia, ascites.	Death by exhaustion.
2	Abraham S.....	45	1900	Anemia, splenic tumor, ascites, dyspnea.	Fatal.
3	John P.....	50	1902	Enlargement of spleen and liver, ascites and anemia.	Fatal.
4	Jos. S.....	38	1903	Anemia, enlargement of spleen and liver, little ascites, achylia.	Unknown.
5	John H. C.....	32	1905	Very la ge spleen, large liver, anemia.	Unknown.
6	Andreas K.....	36	1905	Very severe anemia, enlargement of spleen and liver, ascites; pain in bones, marked anorexia.	Fatal.
7	Jul. C. J.....	43	1906	Very large splenic tumor; liver markedly enlarged, ascites, dyspnea, later icterus.	Unfavorable, still under treatment.

GROUP II.

8	Nicholas W....	40	1896	Great enlargement of spleen and liver, profuse gastric hemorrhages, recurring thrice in four months.	Fatal.
9	Caroline A.....	52	1906	Splenic tumor, liver considerably enlaiged, severe anemia, frequent piofuse intestinal hemorrhages, ascites.	Fatal.
10	Arthur, J. M...	48	1905	Large splenic tumor, large indurated liver; gastric hemorrhages, ascites.	Fatal.

GROUP III.

11	William R.....	58	1894	Large liver, slightly enlarged spleen, anemia, hyperchlorhydria.	Cured.
12	Anton T.....	33	1898	Liver and spleen enlarged, attacks of chills and fever without icterus.	Was impr'v d for three years, then fatal.
13	Louis M. D.....	42	1903	Spleen and liver enlarged, severe anemia.	Cured.
14	Michael M. L. .	48	1904	Spleen and liver enlarged, anemia and slight leucocytosis, dilatation of stomach with beginning pyloric stenosis, ischochymia, hyperchlorhydria.	Much improved.
15	Beni. L.....	34	1905	Spleen and liver enlarged, anemia.	Unknown.
16	August F.....	32	1905	Liver and spleen enlarged, ascites, anemia, hyperchlorhydria.	Improved.
17	Elmer E.....	49	1902 and 1906	Liver and spleen are enlarged and hard, partially lobulated; ascites, dyspnea.	Improved for years.
18	Peter H. K.....	50	1906	Liver and spleen enlarged, anorexia, occasionally vomiting.	Cured.

In most of my cases the liver was considerably enlarged and usually much firmer than normal; changes in the contour of the liver in the shape of round nodules were twice found. Regarding the ascites of the first group my opinion coincides with that of Banti, in so far as the ascites shows absolutely no tendency to absorption, and that it will return quickly when artificially removed. The hemorrhagic form is especially important, as it is often mistaken for ulcer of the stomach.

I would like here to sketch Case M. M., forty-eight years old, a politician, has always lived well, occasionally drank too much, when with friends,

although not a habitul drinker; was attacked in October, 1905, while in the best of health by severe hemorrhage from the stomach. The amount of blood vomited was supposed to have been at least a quart, and an undetermined quantity was lost in the bowel. A week later he felt so well that, contrary to the advice of his physician, he attended a banquet with his friends. He ate everything, but drank very moderately. When he came home, toward midnight, he again vomited more than a quart of blood and became unconscious. The next day the hemorrhage returned.

I first saw the patient two days after the last hemorrhage. On examination a large splenic tumor was found; the liver was much enlarged and firm; a considerable amount of ascites was also present. The patient was then somewhat comatose. Three days later he died after another hemorrhage.

Another case, also belonging to the hemorrhagic variety, is the following one:

Mrs. Q., about fifty-two years old, has been suffering for about six months with diarrhea and repeated intestinal hemorrhages. She lost in weight and became visibly weaker. When I saw the patient I found the following conditions: A pale, well-nourished woman; chest organs normal; abdomen distended; splenic tumor of large size, reaching to a finger's width below the navel, with a smooth surface, and of firm consistency. The liver is considerably enlarged, projecting three to four fingers' width beyond the free margin of the ribs. Ascites can easily be detected; abdomen not sensitive to pressure; rectum and vagina show normal conditions; urine is highly concentrated and contains neither albumin nor sugar. The blood shows severe oligocythemia, also oligochromhemia, but no leucocytosis. The patient died a few days later from an-

other profuse intestinal hemorrhage. An autopsy was not performed, but the case seems clinically to belong to this group; for in carcinoma of the intestines we meet with the enlargement of the liver, but not with the tumor of the spleen of such size.

The third group is especially noteworthy, because a cure may be effected. In this class of cases the enlargement of the liver exceeds that of the spleen, and frequently severe dyspeptic symptoms are found.

It will not be out of place to sketch a few of these cases a little more minutely.

CASE I.—May, 1896. William A. R., fifty-eight years old, has never drank much and has never had any venereal disease or malaria. About a year ago the patient began to complain of lack of appetite, frequent eructations, constipation, and at times a feeling of oppression in the chest. The patient lost a little in weight and became much weaker. Soon dyspnea developed, particularly when walking, and occasionally also after dinner even without any exertion. Physical examination showed: Liver much enlarged and somewhat hard; contour regular; reaches down to the umbilicus. Spleen can also be palpated at least a hand's width below the left costal margin. It feels rather hard and has a smooth surface. The stomach contents show a high degree of hyperacidity; otherwise nothing abnormal. In the blood slight anemia without leucocytosis or plasmodia was found. The urine was free from albumin and sugar. Patient was treated with alkalies without, however, feeling much better. The condition remained about the same for nearly a year, except that the weakness increased. In May, 1897, the patient was compelled to remain abed, suffering from rise in temperature toward evening. He received 0.5 gm. of quinine twice daily, with 0.2 gm. of methylen blue once daily. Later he was

treated with iodide of soda and iron. His condition improved slowly, and in November, 1897, patient could be considered cured. The enlargement of the liver and spleen had disappeared, and the strength of the patient had returned. Patient has been enjoying uninterrupted health ever since and is still alive.

CASE II.—October 21, 1903. Louis M. D., forty-two years old, had always been well until about ten months ago, when he began to suffer from digestive disturbances. The appetite disappeared, and right after dinner usually a feeling of fullness developed. Severe pain was absent, vomiting occurred only rarely, whereas a marked degree of constipation was present. Patient lost steadily in weight, about forty pounds in all, and became exceedingly weak. He has never been drinking to excess and never had syphilis. On examination the liver is found to be considerably enlarged. The dullness commenced at about the fifth rib and reached to the umbilicus. The organ feels harder than normal and has a few superficial irregularities. Spleen also is much enlarged and projects about three fingers' widths beyond the costal margin. Its surface is smooth and its consistency harder than normal. The mucous membranes look pale and anemic. The stomach is not dilated and its contents show the presence of free HCl. in nearly normal quantity. The thoracic organs as well as the urine are normal. The diagnosis of hypertrophic cirrhosis of the liver is made. The patient received iodide of sodium and iron, was put upon a liberal diet, and his bowels were regulated. He improved from day to day, became stronger, and gained in weight. At the end of November, 1903, the boundary of the liver was found to be nearly normal; the spleen also was considerably smaller; it could only be palpated on very deep

inspiration. He had gained forty-five pounds, and remained perfectly well.

CASE III.—December 15, 1903. H. N. B., thirty-eight years old, was suffering for the last three years from frequent pains in the stomach, occurring usually from one to two hours after meals. These were relieved by the taking of food. Since the beginning of 1903 patient complained of fullness after meals and increasing weakness and lost forty pounds in weight. He has never been a heavy drinker, nor has he had syphilis or malaria. Patient is of a somewhat brown but not icteric complexion and looks emaciated and anemic. Examination reveals a marked enlargement of the liver. The dullness commences a finger's width below the right mammilla and reaches down to the navel. The liver is easily palpated, being somewhat harder than normal, but has a perfectly smooth surface. The spleen is only slightly enlarged; the dullness goes to a finger's breadth above the margin of the ribs. In the thorax there is nothing abnormal. Urine is free from sugar or albumin and contains no bile pigment. The blood shows a diminution of the erythrocytes without any marked leucocytosis. The stomach contents revealed marked hyperchlorhydria without any signs of ischochymia. The patient is given alkalies and iodide of sodium with iron. He is also liberally fed. Under this treatment the condition of the patient improved markedly, and two or three months later he was perfectly well. Spleen and liver are apparently perfectly normal, and there is complete euphoria. Two and a half years later I again saw patient. He told me that during that time he had remained perfectly well and had gained fifty pounds.

CASE IV.—June, 1904. Michael L., forty-eight years old, has been suffering for ten years from severe pains in the stomach, accompanied by frequent

vomiting. Food seems to have alleviated the pain. For the last three years he has been using the stomach pump and frequently washes out his stomach in order to ease the pain. In spite of good care and apparently rational treatment, the patient kept losing flesh (twenty pounds) during the last year and became so weak that he had to spend the greater part of his time abed. Fever had evidently not been present, but intense paroxysms of pain in the upper abdominal region occurred, so that injections of morphine were required in order to afford relief. At present he is complaining of pains over the entire abdomen, of pyrosis, constipation, and great weakness. Status presens; thoracic organs are normal. Inspection of the abdomen reveals a protuberance of the upper part, especially toward the right, also peristaltic waves, running from left to right, occupying the region of the left side of the abdomen from the costal margin to the pubis. Clapotage is easily produced in this region. On palpation enlargement of liver and spleen is found. The first organ reaches to a finger's width above the navel, has a rather smooth surface, and feels harder than normal. The hepatic dullness begins a finger's width below the mammilla. The spleen can be felt as a smooth, oval tumor ten centimeters below the free edge of the ribs. It feels rather hard. The stomach is now emptied with the stomach tube and thoroughly irrigated. The contents show food remnants from the previous day, and also a high acidity (100), much HCl, and very many microorganisms (especially yeast cells). The diagnosis was made of a combination of Banti's disease with benign ischochymia caused by a beginning pyloric stenosis. Patient was then treated according to the usual methods for his stomach affection, and he improved from day to day. In September all signs of ischochymia and

dilatation of the stomach had disappeared and hyperchlorhydria alone remained. He had also gained in weight in the meantime. The condition of the liver and spleen was the same. He was, therefore, advised to have his epigastrium treated two to three times weekly with *x*-rays. In January, 1905, the spleen could still be palpated, but it did not project beyond the free margin of the ribs. The liver had also become smaller, projecting two fingers below the ribs. He was given iron and atoxyl. I saw him for the last time in January, 1906. He attended to his business in 1905 and enjoyed good health. Since June, 1904, when I first saw him, he had gained fifty pounds and was free from subjective complaints.

In closely scrutinizing the cases of Group III, just sketched, the following may be said: We have to deal here with an affection in which the liver is considerably enlarged (often twice the normal size). Its surface is usually smooth, but not always. It sometimes has slight irregularities. Its consistency is usually somewhat harder than normal. The spleen is also enlarged; it does not, however, reach the size of that in Groups I and II. Severe stomach affections are frequently found (R, B, L) and seem to influence the course of the disease. A perfect cure frequently results.

It is difficult to classify these cases in any clinically well-defined group. We have to deal here with a hypertrophic process of the liver, which will also become normal under favorable conditions. The whole picture seems to resemble hypertrophic cirrhosis of the liver, if not identical with it. Fatty liver could hardly cause similar symptoms, especially the increase of consistency, which is usually not found in this affection. As we have to deal here besides with an enlargement of the spleen and ane-

mia, I have placed these cases at present under the category of cirrhosis of the liver and splenomebalia, or Banti's disease.

Regarding treatment we must consider with which group and stage of the disease we have to deal. In Group I and II during the preascitic stage extirpation of the spleen might be considered, as first recommended by Banti. If operation is not decided upon, then *x*-ray treatment would be desirable. I have had good results with this treatment in one case of Banti's disease (Michael L.) and in two cases of pseudoleukemia splenica. The splenic tumor was reduced in these cases to such a degree that it could be felt only with difficulty underneath the ribs. This treatment was accompanied by improvement of the other symptoms.

Gastric hemorrhages will have to be treated according to the accepted rules; we may be slightly bolder in giving food in these cases.

In the ascitic stage of the first two groups the prospects of improvement are very bad. The fluid quickly reaccumulates after puncture, and thus the patients gradually approach their death.

It is altogether different in Group III. As mentioned above, we often meet with cures, not only in the beginning of the disease, but also when the symptoms have advanced.

It is essential to combat the stomach symptoms and to put the patient on a strengthening diet. I have also used sodium iodide in conjunction with iron, and sometimes arsenic, and usually obtained good results.

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DISCUSSION.

DR. EINHORN: Before reading my paper, I shall take the liberty of demonstrating to you the method which I described not long ago of testing the digestive functions by means of beads which are covered with certain food substances. The object of this test is to see not only what the stomach does, as we learn from an analysis of the stomach contents, but also to give us an idea of what is accomplished in the way of digestion in the intestinal canal.

The original method which I employed was found to be correct, but rather too complicated. It consisted of administering to the individual a number of beads in a capsule These beads were coverd with certain food substances, such as thymus gland, meat, catgut, mutton fat, potato, etc. The beads were afterwards searched for in the stools of the individual who had partaken of the capsule. In persons with normal digestion they were usually recovered in the first or second stool. In crtain cases, however, some of the beads were not passed for eight, nine or ten days, and unless the examination of the stools was very thorough, they were easily lost entirely. This constituted a serious objection to the method, and one day, while searching for the beads, the idea suddenly occurred to me that instead of giving them separately in a capsule, I could tie the six of them together, and administer them in that form in the capsule, and recover them all together. Another modification I adopted was to give a mild cathartic, usually castor oil, to expedite the expulsion of the beads if they were not passed voluntarily within twenty-four hours after their administration. By these modifications, the test has been greatly simplified. Here is a digestive bead string that is ready for use. These beads are covered with various food substances and put into a capsule, and given to the patient after a meal, and then the stools are carefully examined until they are recovered. I also pass around for your inspection several beads which were recovered from the stools. Here is one that was covered with unpeeled potato; nothing is left but the skin of the potato, showing that the individual's digestion of starchy food was good.

I think if we make tests of this kind in a great many individuals, they will in time furnish us with valuable data regarding the digestive functions. I have had only a few cases where the various food substances on the different beads remained entirely unchanged by their passage through the digestive tract. One was a case of achylia gastrica; the other I described in my original paper on this subject, which appeared in the Medical Record of February 10th, 1906.

The examination of the stools in order to recover the beads is very simple, requiring nothing more than a stool-sieve. The beads are usually expelled in the course of 24, 36 or 48 hours, or, if you are in a hurry, their expulsion can be expedited by a dose of castor oil.

DR. J. KAUFMANN, OF NEW YORK: I think the symptom complex given by Banti is at best not a very well defined

one, and we should be very careful about applying it to cases, even if they do show some of the symptoms or a combination of the symptoms named by him.

The only time that I was inclined to make the diagnosis of Banti's disease, and especially of the hæmorrhagic type, was in a patient who had been suffering for some years from a severe type of malaria which he had acquired in Honduras. Both the liver and spleen were much enlarged, and he had suffered from hæmorrhages from the mouth and nose, and subsequently a very severe one from the stomach. The case ended fatally, and the autopsy showed a chronic gastric ulcer, about the size of the palm of an adult hand, which was doubtless the source of his gastric hæmorrhage. The enlargement of the liver and spleen were probably the result of his chronic malarial poisoning, and yet the whole picture in this case would have led one to believe that it was one of Banti's disease.

Dr. I. Adler, of New York: I agree entirely with Dr. Kauffmann that before making the diagnosis of Banti's disease, the complete syndrome must be present. It is perfectly well known that there are many forms of cirrhosis of the liver, both atrophic and hypertrophic, associated with enlarged spleen and ascites, and very frequently with hyperchlorhydria, and which eventually give rise to various types of anaemia. There is another group of cases which are usually benefitted by the iodides, and in those a history of syphilis is usually obtainable.

I would like to call to your mind the fact that Marchand, of Leipsic, has made the claim that Banti's disease is not a disease *sui generis*, but is of syphilitic origin in every instance. In that connection I might mention a case of supposed Banti's disease which proved to be one of gumma of the liver and recovered entirely under the use of potassium iodide.

True atrophic cirrhosis of the liver may be associated with old or recent gastric ulcer, with hæmorrhages, and the symptom complex in such a case might easily mislead one into making the diagnosis of Banti's disease.

It should be remembered that in Banti's Disease there is a *primary* enlargement of the spleen besides a rather peculiar blood picture.

Dr. A. L. Benedict, of Buffalo: The very fact that the so-called Banti's disease is so rare militates against its being a clinical entity, because, according to the laws of chance, any symptom complex that is a clinical entity and not a mere coincidence must occur fairly frequently.

In connection with the diagonsis of Banti's disease, I would like to raise the following queries. How is it known that the splenic cirrhosis is primary, that there is no cirrhosis of the liver which has thus far not produced any change in the size of the organ? We may happen to examine the patient during a period of temporary congestion, when the consequent temporary increase in the size of the liver, compensates for a cirrhotic shrinking. I recall seeing an operation lasting about three-quarters of an hour, during which brief period an apparently much enlarged liver

became reduced in size by a hand's breadth, the enlargement having been due to congestion.

In connection with the demonstration given by Dr. Einhorn, I would like to ask him whether he has had any difficulty in getting capsules that are perfectly soluble? Personally, I have found that the ordinary capsule will fail to dissolve in water after an hour, or sometimes an entire day and will, sometimes, pass the anus entire.

Dr. J. C. Hemmeter, of Baltimore: The most extensive account of the Banti syndrome is by Osler, who I believe has reported the largest number of cases. Dock, of Ann Arbor, has also described a number of cases, and given the histological changes, with drawings.

A singular fact in connection with the primary splenomegaly is that in two of Osler's cases removal of the spleen cured the patients. The operations were done by Halsted, and were reported in the Johns Hopkins Hospital Bulletin. One has only to read Osler's paper to see what haziness still exists in regard to this snydrome. I was told by Dr. McCrea of Baltimore, that during an interview he had with Banti, the latter informed him that he had read Osler's paper and it had convinced him that Osler did not understand his disease at all. That he (Osler) had mistaken something else for the syndrome that Banti had described. This seemed to indicate that further pathological investigations were necessary before we could arrive at a clear idea of this symptom complex.

I have seen one case which I regarded as an example of Banti's disease, and in which Dr. Harry Adler, of Baltimore, made the blood examinations. Antisyphilitic treatment was given for a time, but did not do any good. The patient ultimately died, and a histological examination of the liver gave findings which corresponded very closely with those of Dock.

Dr. Einhorn: In my paper I reported a group of cases that presented certain symptoms. Whether we call them Banti's disease or somthing else is immaterial. In Banti's own description, he included only those cases in which there was a benign tumor of the spleen of enormous size, together with enlargement of the liver, anaemia, and ascites. The cases ended fatally, unless the spleen was extirpated in the primary stage. Senator, in the Berliner Klinische Wochenschrift, includes under this head cases of splenomegalia of the Hodgkin's variety, and Von Aldor has described cases of cirrhosis of the liver, with immense enlargement of the spleen, and accompanied by hæmorrhages, which he also regards as a variety of Banti's disease

The prominent symptoms in this syndrome are a cirrhotic process in the liver, enlargement of the spleen, and anaemia. The latter symptom might not be of a very intense type, and differs entirely from pernicious anaemia. The reduction in the percentage of hæmaglobin is out of proportion to the loss of red blood cells, which is directly opposed to what we find in pernicious anaemia. Furthermore, the change in the appearance of the red blood corpuscles does not tally with that observed in pernicious

anaemia. There is also a diminution in the number of white blood corpuscles, a condition known as leucopenia.

I wish to emphasize that clinically we have a third group of cases which are more or less similar in regard to the condition of the liver and spleen, and which have a tendency to recover. I have made quite a number of observations on syphilis of the liver and spleen, and regard those cases of an entirely different type from Banti's disease. The spleen is not so much enlarged, being very little if at all larger than normal, and the liver presents peculiar characteristics, its surface being covered with tumefactions. In Banti's disease the surface of the liver may also be hardened, but these tumefactions are not so distinct and do not disappear so quickly under the use of potassium iodide. In the cases reported in my paper, there were no symptoms nor evidences of syphilis. The cases were of an entirely different type from those of syphilitic origin.

With regard to malaria, Banti himself reported cases in which malaria had been present in former years, but in my cases there was no history of malaria, and no plasmodia malariae were found in the blood. Anaemia was always present, but not very pronounced at first.

In reply to Dr. Benedict's question I would say that the capsules I use are supplied Eimer & Amend, of New York City; they have proven entirely satisfactory, and I have never known of an instance where they failed to dissolve.

In reply to Dr. Spivak as to the average time it takes to recover the beads, I would say that there was no definite rule in this respect. The shortest time it took some of the beads to pass through the gastro-intestinal tract was four and a half hours, and the longest time was nine days.

THE REFLEXES CONTROLLING THE PERISTALTIC MOVEMENTS OF THE OESOPHAGUS.

By Dr. S. J. Meltzer, of New York City.

DISCUSSION.

Dr. J. C. Hemmeter: I wish to congratulate the members of the Association for the opportunity they have enjoyed of listening to this interesting physiological investigaton. In the experimental work that has been done regarding the peristaltic action of the oesophagus, Meltzer's name stands as a pioneer. His investigations on the subject are referred to in almost every text-book of today, including that of Ludwig & Haidenhein, while in Luciani's magnificent work, an entire chapter is devoted to the results of Meltzer's experiments.

I would like to ask Dr. Meltzer if he believes in selective peristalsis of the oesophagus; that is, a different peristaltic action in different localities, and also for solids and fluids, as Cannon has recently brought out in his X-ray studies?

Dr. Meltzer: In reply to Dr. Hemmeter's question as to whether there was a selective peristaltic action of the oesophagus, I would say no. The body had two methods of conveying the contents of the mouth to the stomach. One was by throwing them down by the force accumulated in the mouth, and the second was by peristalsis. Whether the methods proved successful, or, whether both methods are active in each special case must be decided by a number of different factors, including the size and the consistency of the morsel to be swallowed, and also the condition of the oesophagus.

ACHYLIA GASTRICA.*

BY FRANKLIN W. WHITE, M.D.,
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ACHYLIA gastrica is a term introduced by Einhorn to denote an absence of gastric secretion, lasting over a greater or less interval of time. The gastric contents contain no free or combined HCl, and the ferments are absent, or reduced to insufficient and unimportant traces. The condition is not rare, though the literature of the subject is rather limited, and an accurate knowledge of the condition is largely confined to specialists.

Achylia is a sign of disturbed function of the stomach, which may accompany various disease conditions, such as cancer, severe anemia, and gastric catarrh, and the evidence of recent years tends to show that it also occurs frequently apart from primary organic disease of the stomach or other organs, as a purely functional disturbance.

The history of achylia is closely associated with that of atrophy of the gastric mucosa, and the discovery of a lack of gastric secretion in cancer, and atrophy, led to the false opinion that if no HCl or ferments were found the diagnosis of cancer or atrophy was justified; and the association of achylia with severe and fatal disease led also to the false belief that it was necessarily a severe condition leading in a short time to

* Read by invitation of the American Gastro-Enterological Association, at the ninth annual meeting, Boston, 1906.

death. We know now that an absolute and permanent lack of gastric secretion is compatible with health and happiness, and that the gastric secretion can be spared and compensated for by intestinal digestion. In short, we can make a sharp distinction clinically between (*a*) secondary achylia associated with cancer, severe anemia, or atrophy of the gastric mucosa, and (*b*) simple achylia without atrophy, which is a benign condition.

The cases which I wish to report were chosen from the following material: In 235 consecutive cases of chronic disease of the digestive organs which I have recently seen, there were 183 cases of disease of the stomach, consisting of neuroses, ulcer, cancer, catarrh, dilatation, etc., in which the ordinary analysis of the stomach contents was made. In this number, there were 41 cases of subacidity, not including cases of cancer of the stomach or other organs, and in the group of subacid cases were 9 patients in whom there was either entire or almost entire absence of all elements of the gastric juice,— a condition of achylia gastrica. In two of these patients, the condition is undoubtedly the terminal stage of a chronic gastric catarrh; they are only briefly mentioned in this paper; in the other seven, the condition is apparently one of simple primary achylia gastrica.

I have been testing the gastric contents for ferments in all cases in which free HCl was absent after a test meal and the total acidity very low, and in this manner these cases of achylia were recognized. I believe this condition would be more frequently found if it were carefully looked for.

These cases form an important and interesting group,— important, because of their considerable

frequency, and the resemblance of some of them to gastric cancer. Several of the patients came to me with a diagnosis of cancer, and in one a laparotomy was performed, as a result of this diagnosis made by another physician. The patients are all men, whose ages varied from twenty-seven to seventy-three years. They have been under observation for a period varying from ten months to several years, with the exception of two undoubted recent cases in young men, which have been followed only three months. The diagnosis of achylia gastrica has been made from the results of examination of the stomach contents after a test meal in connection with the clinical history and physical examination of the patients.

Method of examination. — Each patient had a thorough physical examination, and the stomach contents, obtained by expression or aspiration three-quarters to one hour after an Ewald test meal was examined as follows: The amount of contents, its appearance, whether well digested or not, and the amount of mucus were noted. The test for free HCl, was made with Gunzburg's reagent, and the total acidity estimated, using phenalphthalein as an indicator. The combined HCl was tested for occasionally by the qualitative Sjoquist-Ewald method,[1] but where the total acidity was five or less, in other words, no more than that of the test meal which had been eaten, the test for combined HCl was considered unnecessary.

The test for lactic acid was made by Uffelman's simple method, and if positive, confirmed by

[1] To ten cc. of filtrate add barium carbonate, evaporate to dryness and reduce to ash. Dissolve the residue in water. If, on addition of a solution of sodium carbonate, no precipitate is found, it shows the absence of barium chloride and that no combined HCl was present.

repeating the test with an acid ether extract of the gastric contents. The test for pepsin and pepsinogen was made by the Hammerschlag method. Occasionally no gastric contents could be obtained one hour after the Ewald test meal, in which case the test was repeated and the contents aspirated at the end of three-fourths or one-half hour. The test for rennin and renninzymogen was made by Boas' dilution method. Peptones were tested for by the biuret reaction. In several cases, fragments of mucous membrane were found, which were carefully examined. The examination of the stomach contents was repeated many times in each case.

The stools were examined according to the Schmidt-Strassburger method, especial attention being paid to signs of imperfect gastric digestion, such as residue of connective tissue and muscle fiber. The guaiac test for invisible blood was applied to the acetic acid-ether extract of the stools in each case.

Clinical history. — I will summarize the clinical history of these patients, first speaking briefly for purposes of comparison of the two cases in which the achylia was evidently a sequel to chronic gastric catarrh.

The ages were forty-three and seventy-five. Both had used alcohol and tobacco to excess, and one was an excessive tea drinker. The duration of gastric symptoms was five and ten years respectively. These consisted of distress after meals, especially when hard food was eaten, anorexia, frequent vomiting of slimy stuff, gradual loss of 10–20 lbs. weight, slight anemia, occasional mild attacks of diarrhea. Physical examination showed nothing abnormal save slight anemia, and malnutrition. The size and position of the stomach were normal. The contents of the fasting stomach

consisted of 10–20 cc. thick ropy mucus containing considerable epithelium and leucocytes. The residue after the test breakfast was similar to the cases next to be described. The stools in the older man were thin fluid and foul during attacks of diarrhea, and then and also at other times showed microscopically many well preserved muscle fibers, and considerable connective tissue in small flakes. The stools of the younger man were normal.

The ages of the cases of simple achylia gastrica were as follows: Three of them twenty-seven, the others thirty-three, thirty-six, forty-three and seventy-three years. Four were markedly neurasthenic. The course of the illness was variable, and the symptoms were none or many. In one case, there were at first no symptoms referable to either the stomach or bowel, and the examination of the gastric contents was made in searching for an explanation of debility and loss of weight. In one case, there were no gastric symptoms, but the patient came for treatment of diarrhea, which evidently had its source in the stomach. Oppler, Einhorn and Schmidt have pointed out the relations between achylia gastrica and intestinal catarrhs. In four cases there were gastric symptoms, such as distress after meals, belching of gas, anorexia, also many nervous symptoms, associated with moderate loss of weight, and in two of them occasional attacks of diarrhea. All these patients made some permanent gain in weight under treatment, three of them from 3 to 5 lbs., one 8 lbs. and one 15 lbs. In none of the patients were the symptoms severe, or even very troublesome. Intervals of comparative health or entire absence of symptoms alternated with relapses due to overindulgence in food, or the result of the weather.

Two of the patients when last seen had had no trouble with stomach or bowel for months. They illustrate well the entire lack of symptoms in achylia when the motility of the stomach is not affected, and the bowel not irritated. The intestine does the work of the stomach, if there is no stasis of food, and no lack of nutrition results. The patients feel perfectly well, and either maintain their weight, or gain materially.

The ultimate course of the achylia is interesting. The gastric secretion in the two patients with chronic atrophic catarrh showed no change for the better, as might be expected. In six cases of simple achylia, the condition has persisted up to the present time; in four, over a period of observation of one to two years, and in two cases for two months.

In one case a return of gastric secretion to approximate normal conditions has occurred. This was a nervous, over-worked student of twenty-seven years, who had suffered for the last seven years with distress two hours after meals, with occasional regurgitation of food. He was first seen nearly a year ago, when the stomach contents after a test meal showed no free HCl, total acidity 3, and absence of ferments and their zymogens. This condition persisted unchanged for five months under observation, at the end of which time the gastric secretion was found to be entirely normal, free HCl 25, total acidity 41, ferments normal. This normal condition has continued without variation up to the present time. No definite explanation of the change can be given. The patient's general condition has considerably improved under rest, careful diet and tonics, but headaches and other nervous symptoms persist. There has been a gain in weight of three pounds only.

Physical signs. — Thorough physical examination of these patients showed no other bodily ailment to which the conditions of the stomach were secondary. One was a slender, overworked student, three were vigorous young men, and three were older men, whose loss of weight, slight anemia and debility made it necessary to consider the possibility of incipient gastric cancer.

Examination of the blood revealed a normal condition, or slight anemia. The red cells varied from 4,500,000 to 5,400,000; the white cells from 7,000 to 8,000 and the hemoglobin from 70% to 100%.

The stomach was found to be of normal size and position in all. Examination of the contents of the fasting stomach showed no signs of chronic catarrh or stasis of food. The characteristic features of the stomach contents were the small amount, little fluid, undigested appearance, absence of free and combined HCl, the complete or almost complete inability to digest proteids when rendered mildly acid (0.20%) with HCl, and the complete or almost complete absence of power to coagulate milk. The amount of gastric contents was small (10 to 50 cc.), often so small that it was necessary to aspirate the contents in less than an hour in order to obtain any residue; this may be due either to the rapid emptying of the stomach, or to the fact that the stomach had lost its power of secretion, and of diluting the gastric contents, or to the thick pasty nature of the residue, which was difficult to aspirate through a tube.

The gastric contents looked like food which had been simply chewed and swallowed, and not digested. No excess of mucus was present, and the microscopic examination of the sediment

showed nothing characteristic except fresh blood corpuscles. The filtrate of the stomach contents showed no free HCl, and a total acidity of from 2 to 5, no greater, in fact, than that of the food taken (the acidity of an Ewald test breakfast of 35 gm. of bread well macerated in 300 cc. of water, averages 4 to 6. The mild alkalinity of the saliva which is mixed with it probably has a slight neutralizing effect). Pepsin and pepsinogen were invariably absent in 4 cases, and present in minute traces in 3. Rennet and rennet zymogen were always absent in three cases, and in 4 gave a positive result in the undiluted stomach contents, but negative in dilutions. Lactic acid was absent. No biuret reaction for peptone was obtained.

The work of Hammerschlag, Troller, Gintl and Schiff has shown that the secretion of HCl and of ferments in the stomach is more or less independent and that either one may be diminished without the other being affected; and Riegel and others have considered the secretion of pepsin more constant than the HCl function of the stomach. Cases of simple achylia seem to be an exception to the rule, for we have found the ferments very slightly if any more persistent than the HCl. Robin and Schiff have shown that the same conditions may occur in gastric cancer, so that the ferments cannot be used to distinguish simple achylia from cancer.

If we are correct in considering the loss of secretion in our cases to be functional, it is evident that simple achylia forms an exception to the rule laid down by Boas and Cohnheim, that in anacid neuroses of the stomach, the ferment secretion is usually well preserved, while in organic diseases of the stomach, such as catarrhal gastritis, interstitial gastritis and atrophy

of the gastric glands, the ferments are diminished or absent. It is evident that the estimation of gastric ferments cannot be relied upon to distinguish neuroses from organic disease of the stomach.

The gastric mucosa was very easily wounded in getting the residue from the test meal, even when a tube with a closed end and side openings was used and the contents expressed instead of aspirated. Fine blood streaks were occasionally seen, and in three instances, small fragments of the gastric mucosa were obtained. These were carefully prepared and examined. Two showed nothing abnormal. One showed a slight degree of granular gastritis. These findings are interesting, but do not necessarily indicate the condition of the stomach as a whole.

The stools were characteristic. In the absence of diarrhea they showed no gross residue or mucus; fat and starch digestion were normal; microscopically an excess of well preserved muscle fiber was often found, and considerable connective tissue in threads and flakes; this occasionally appeared as a fine network through an otherwise normal stool. At other times, the stools were entirely normal. This is in accord with the results of Von Noorden and others, who have shown that digestion may be perfect in achylia.

When diarrhea occurred the stools were light colored, watery, usually foul, mucus was present often with leucocytes, and considerable epithelium and an excess of fatty acids and soaps, and muscle fibers and even connective tissue appeared as a macroscopic residue and as a fine network through the whole stool. This condition clearly shows a lack of gastric secretion, for we know that connective tissue, if not thoroughly cooked

through, is only or chiefly digested in the stomach. This "connective tissue reaction" is both delicate and useful; it is the simplest and best sign of the bowel trouble which has its source in the stomach.

The presence of macroscopic meat residue in the feces indicates a disturbance of the intestine also, which was no doubt secondary. We know that the stomach does only part of the work of digesting meat, the greater part is done in the bowels. The hypermotility of the stomach may have also played a part in irritating the bowel by rapidly depositing the food there in an undigested condition. It is evident that the lack of gastric digestion produced a great diversity of results in the bowels, varying from slight functional changes to quite severe catarrh. In several instances, the bowel trouble was so pronounced as to entirely overshadow the stomach symptoms. The presence of connective tissue in the stool was of great value in such cases in indicating the source of the trouble.

The repeated examination of the stool for invisible blood by the guaiac method always gave a negative result, which is important in distinguishing these cases from cancer.

Diagnosis. — We have to distinguish the primary symptomatic achylia, the primary weakness of gastric secretion, from the loss of secretion due to the gastric atrophy of cancer, severe anemia or late chronic catarrh.

The primary cases are characterized by the absence of gastric stasis, the absence of mucus, the small residue from the test breakfast and its chemical character, absent HCl ferments absent or in traces, lactic acid absent or in traces the vulnerable mucosa, and the absence of invisible blood in the stools. (The physical signs of

chronic gastric catarrh or cancer occasionally resemble these closely.) The symptoms are much less characteristic; the nutrition suffers no important change. The patients may be young and vigorous, or neurasthenic and delicate or of middle age and beyond.

The diagnosis is most easily made where a total lack of gastric secretion is found in a patient who is young and vigorous, and whose symptoms are few and of short duration. Where no gastric secretion is found in a middle aged or elderly person who has lost flesh, is weak and somewhat anemic, the diagnosis requires careful and persistent study of the case, and may be very difficult. It is imperative to make a diagnosis between simple achylia and cancer in order to give the patient the benefit of appropriate early treatment, and it is important also to distinguish functional absence of secretion from chronic catarrh. There is often a striking clinical contrast between cases of simple achylia and atrophy, the first young, vigorous and healthy; the second old, weak and suffering from severe anemia.

In simple achylia no true cachexia is present, though loss of weight is common because the patient may eat too little. A long history of gastric symptoms and a permanent gain in weight and strength practically exclude cancer. In simple achylia we do not have occult blood in the stools in the absence of other lesions of the digestive canal. In cancer the permanent and abundant presence of occult blood in the stools is the rule. In cancer and in chronic catarrh the acidity of the stomach contents is usually decidedly higher than in simple achylia.

The examination of the fasting stomach is one of the most important means of diagnosis; this is empty in simple achylia while in cancer dimin-

ished motility is very much more common, food remains are frequently found, and lactic acid appears early and constantly. The presence of changed blood, mucus, epithelium and leucocytes favor cancer and catarrh.

Martius says that if achylia persists for months without stagnation and fermentation, cancer may be excluded. This is not absolutely true. Cases of well developed cancer of the lesser curvature of the stomach without stasis, or the *early stage* of any gastric cancer may present this picture, and are peculiarly difficult of diagnosis. The cancer may be latent for a long time, and have only a slight effect upon the general health as long as the motor function of the stomach is preserved and the bowel is normal. We rarely find cases, however, which cannot be diagnosed after close persistent observation.

The vulnerability of the gastric mucosa is not a distinctive feature of simple achylia. Fine streaks of blood and occasional shreds of mucosa are found in the gastric contents in all organic diseases of the stomach, in association with antacidity according to Cohnheim and Kuttner, though perhaps more frequently, more abundantly and as an earlier sign in cancer than in any other condition. Fragments of mucosa dislodged by the stomach tube tell us little about the stomach as a whole and have little value in diagnosis except in occasional lucky instances.

Cases of chronic gastric catarrh are often easily distinguished from simple achylia by the presence of an exciting cause,—misuse of alcohol, tobacco or cathartics, hasty and irregular meals, etc., the long duration of symptoms, the frequent appearance of mucus, the numerous epithelial cells in the stomach contents and wash-water.

The question of exploratory laparotomy for

diagnosis is a very important one. We have seen that most cases of achylia, especially in young persons, present no great difficulties in diagnosis, but there is a group of cases in middle life and old age, where the suspicion of incipient gastric cancer is strong, where it is not wise to delay the diagnosis long lest the time for satisfactory treatment shall pass by. The necessity for early diagnosis in cancer is self-evident, and the question of exploratory laparotomy for the purpose is very important. The surgeons have blamed the physicians for keeping such cases *too long* under observation in attempt at diagnosis, and insist that every case where cancer is suspected should have an early surgical diagnosis in the belief that it is better to open many normal abdomens than to fail to discover and treat a gastric cancer before it reaches an inoperable stage.

On the other hand, we must not forget that even in recent years an exploratory laparotomy, except in especially selected cases, is an operation which carries a very decided immediate risk to life and it tends also to make the physician superficial in observation, because he knows that the diagnosis will be made at operation. I have seen a considerable number of laparotomies done in the last few years in which no lesion of the stomach was found, and not infrequently conditions were discovered which might have been diagnosed without operation. Occasionally the exploratory operation has been followed by a most unhappy fatal result.

I am a firm believer in the value of exploratory laparotomy for the purpose of diagnosis, but it should only be performed in carefully chosen cases, and after the patient has been, for several weeks at least, under constant expert medical

supervision. In three of these cases, aged forty-three, forty-six and seventy-three, an exploratory operation was considered, but not advised, as the diagnosis became reasonably clear without it, and the subsequent history of the patients has justified this course. One of the patients, aged seventy-five, in which a diagnosis of achylia secondary to chronic catarrh had been made, was seen by another physician who made the diagnosis of gastric cancer, and advised a laparotomy. At operation no gross lesion of the stomach was found. The patient, I am happy to say, recovered promptly from the operation and is as well as before.

Prognosis. — The prognosis in cases of simple achylia is good regarding life. The bowel may do the work of the stomach and the patient be free from symptoms and gain weight under careful treatment. My patients gained from three to fifteen pounds each. Ewald reports a case which gained forty-two pounds.

The fact that patients with achylia may remain in good physical condition so long as the stomach empties itself properly and no opportunity occurs for local fermentation of food, does not indicate that the stomach is an unnecessary organ and serves mainly as a reservoir for food. Such persons are rather in a state of unstable equilibrium, which may be easily upset. Achylia is a disease, not an anomaly, and persons who have it are much more sensitive than normal persons to gastric stimuli and irritants. If there is intestinal indigestion also the prognosis may be serious.

The fact that patients who have had achylia for years may in rare instances recover normal secretion should teach us never to give up hope in the individual case, but I see no way at present of telling in advance in which patient this is

likely to occur, though the literature and my own experience indicate that such a change is confined to the neurotic cases.

Treatment. — The indications are to treat the general condition, the neurasthenia and anemia by appropriate measures, and secondarily care for the digestion. In treating the stomach, we try to do three things: (1) To stimulate and re-establish gastric secretion; (2) to compensate for its loss by artificially aiding digestion in those cases in which the pancreas is unable to do all the work; (3) to protect the motor functions of the stomach and avoid stagnation and fermentation of food.

I consider the last two indications much more important than the first. I have had no success with measures aiming to re-establish secretion, and in reading over the reported cases, now considerable in number, in which normal secretion has appeared after months or years of absence, it is not evident that the treatment has had much to do with the result.

The remedies chiefly used for this purpose are HCl bitters, stimulant foods, the stomach douche and electricity. HCl medication is usually useless, though we all know patients who, as Martius says, take 5 to 10 drops of officinal HCl in water after meals and insist that they feel badly without it and with it have no indigestion. It is difficult to see how this dose can have any value apart from auto-suggestion; 10 or 15 drops in water before, during and after meals may accomplish more. Biedert who was himself afflicted with achylia gastrica took 120 drops of dilute HCl daily. Chittenden has pointed out that 2½ liters of 0.2% HCl will be needed daily with a consumption of 100 gm. of proteid and yet none will be left over as free acid which

is necessary for the action of pepsin. Given without pepsin it has no value as a digestant in the stomach, and with pepsin it is useless because not enough can be given to leave free acid. It is useless as an antiseptic, a stimulant to pancreatic secretion, or stimulant to gastric motility for the same reason, and fortunately it is not needed for the latter purpose as stasis is rare in simple achylia. HCl medication has not proved useful or necessary in my hands; the patients have remained well without it, and have recovered from acute attacks of indigestion without it.

If there are signs that the pancreas cannot fulfill the demands made upon it, it is more useful to remodel the diet, or to give pancreatic extract or other ferments active in the bowel. These cases without HCl seem to be the ideal ones for the use of pancreatin. In most cases diet without drugs is sufficient. We know that the pancreas usually compensates for the lack of gastric juice and the discovery of achylia in a comparatively healthy person is not an indication for the use of drugs calculated to stimulate secretion. A perfectly compensated digestive apparatus needs them no more than the compensated heart needs digitalis. A careful regulation of the diet has proved the most important part of the treatment. In selecting the diet it is more important to choose food which will favor the mechanical work of emptying the stomach, and will leave it readily than to seek out those suited to the lack of secretion. This means food so finely divided that it will go through a sieve, brothy foods, vegetable purées, finely chopped meat, not a bulky diet. Butter is very useful. Fine division of the food by chopping, cooking and chewing also lessens the work of the bowel. The patient must eat. He has perhaps decided

that one or another food disagrees with him. One doctor has forbidden this food and another that until he eats far too little. The main thing with ill-nourished patients is to feed them well.

Adolph Schmidt has pointed out that the connective tissue diarrhea of achylia has given us a valuable indication for the treatment of the intestinal disturbance. The absence of the antiseptic action of HCl of the stomach has been considered the cause of these bowel troubles, but the HCl is only a factor in keeping down the fermentation, for two thirds of the patients with achylia have no bowel trouble. In general, lessened motility of the stomach has greater significance as a cause of bowel trouble than disturbances of secretion. When food leaves the stomach slowly, germs of fermentation and putrefaction establish themselves there more easily, and are emptied into the bowel, and in this fashion cause secondary trouble. This, however, is not important in simple achylia as stasis of food in the stomach very rarely occurs. The most important factor is a lack of digestion of connective tissue in the stomach. The bowel cannot digest raw or smoked connective tissue, and if this is not digested before leaving the stomach, it goes through the bowel practically unchanged. Gastric digestion of meat, of fat and of starch is not so important, because here the bowel secretion may compensate for it in a general diet. Where meat is not finely divided it is not only the connective tissue which is lost when the stomach does not do its work, but also the inclosed muscle and fat. Pieces of meat and fat may appear in the stool. This overloading the bowel with undigested material is very important as a cause of diarrhea and finally of catarrh of the bowel.

The number of intestinal bacteria depends largely on the quantity of food at their disposal. The better the food is digested, the fewer the intestinal bacteria. Many cases of diarrhea are undoubtedly explained by the growth of germs of fermentation and putrefaction in poorly digested food residue. If the patient over-eats he gets a diarrhea, not because he has swallowed too many bacteria, but because he has too much undigested food in the bowel which serves as a culture media for these omnipresent germs. No intestinal antiseptic or cathartic has any marked effect on the number of intestinal bacteria. Their number can be kept down only by taking away their food.

The indications for treatment to be drawn from this are briefly that we must not give patients with achylia raw, rare or smoked meat. We have seen that the bowel is unable to digest raw connective tissue and we must exclude it unless it can be shown that the use of raw and smoked meat has definite advantages of another sort over well-cooked meat. The experiments of Pawlow have shown that the use of raw flesh stimulates the secretion of the digestive fluids which is especially important in gastric antacidity. If we compare, however, the advantage of stimulating secretion with the disadvantage of a large amount of indigestible connective tissue, we find the latter is most important, and secretion can be equally well stimulated by means of meat extract, bouillon, etc.

Etiology. — We can only briefly consider the etiology of this interesting condition. It used to be generally assumed that achylia gastrica developed only after severe organic disease; and the atrophy of the gastric glands associated with cachexias, cancer of the breast, pernicious anemia,

etc., has been carefully studied post-mortem by Fenwick and others. Since the introduction of the stomach tube cases of atrophy which were associated with pernicious anemia, cancer, senility and the terminal stage of chronic gastritis have been diagnosed during life. We have seen, however, that there is a large group of cases in which the achylia is not associated with these severe diseases, and does not threaten life, and which consequently have been very little studied in the autopsy room. In these there is no history of gastritis or other definite cause. They are apparently chiefly functional cases in which the glandular elements are not usually greatly altered, though they do probably change after long disuse. Analogy is seen in the kidney where the function may be suspended without atrophy of the organ, and in the atony of the stomach and bowel without organic change seen so frequently and persistently in neurasthenia and hysteria.

The etiology of this simple achylia gastrica is an interesting biological problem with a practical side. A well-known and important function is absent without definite cause, or apparent results. The latter may be explained by compensation. The intestinal digestion is so powerful that gastric digestion seems almost superfluous. It is not, however, simply an anomaly, the disappearance of a function which is unnecessary for the organism, (*cf.* Darwin), it is a diseased condition. The individual with achylia is worse off than his mates, — he is in a state of unstable equilibrium, and bowel trouble may be a real danger. The fact that achylias are neurasthenic is not a sufficient reason to consider this the cause. We find neurasthenia without loss of secretion, and loss of secretion without neurasthenia.

Martius considers it a primary weakness of secretion which is either congenital or develops on the basis of a predisposition. The hereditary element is interesting. Cases have been reported in a mother and daughter, in two brothers, etc. An anatomical basis for the condition has been suggested. Hemmeter cites ten cases of achylia which do not include cases of cancer or chronic gastric catarrh, in nine of which fragments of the mucosa obtained through the tube were examined. These all showed signs of granular gastritis and atrophy. Hemmeter questions whether the condition is ever a neurosis. The changes in the mucosa, however, seem far too slight to explain the striking change in function (compare the conditions in gastric cancer where the secondary change in the mucosa leads to achylia only when it has advanced to atrophy), and the granular gastritis may be the result not the cause of the achylia. It is not satisfactory to use the evidence from fragments of mucosa, obtained through the stomach tube, to settle a question of secretion which is the expression of the function of the whole stomach. In addition we have seen one case similar to those reported by Einhorn and others, of recovery of gastric secretion after a long period of achylia showing that achylia may exist when the glands of the stomach have not been totally destroyed (as shown by their recovery of secretion). In such cases the condition is probably due to nervous influences with partial atrophy, but no autopsies have been reported and the atrophy can only be conjectured, not proved. In short, cases of achylia may be divided into two groups: First, the severe and striking cases which were earliest reported in which there is unquestioned atrophy of the mucous membrane (either idiopathic or

secondary to some serious condition such as cancer of pernicious anemia), and second, the group of cases which are by no means rare, in which achylia occurs in neurasthenic weak persons, those of middle life and beyond, where the condition is simply that of a weak stomach with feeble secretion.

In conclusion, I wish to emphasize these points:

(1) Simple achylia gastrica is a relatively common condition. It made up nearly 5% of this group of cases.

(2) The diagnosis must be made from the physical examination of the patient and his stomach contents and feces. The symptoms are not characteristic.

(3) Cases in which there is entire lack of HCl secretion, but in which very minute traces of pepsin or rennin are retained are not achylia in the strictest sense of the word, but are best classified as achylia.

(4) In making the diagnosis of gastric cancer, remember the frequency of simple achylia, which may resemble it closely. The examination of the feces for invisible blood is especially useful here.

(5) The etiology of simple achylia is unproved; its course suggests a functional origin.

(6) Its most satisfactory treatment is hygienic and dietary..

Condensed histories of the seven cases of simple achylia gastrica follow:

Case I. Age twenty-seven. Student. Pleurisy ten years ago. No tobacco or alcohol. Very nervous and overworked. For seven years distress two hours after meals, now all the time. Regurgitation of food. Headache. Weakness, dizziness, appetite good, bowel regular, no vomiting. Best weight two years ago 136; now 121. Objective examination: Slender and rather

thin. Hemoglobin 85%. Reds 4,800,000. Whites 8,000. *Stomach:* Fasting contents, 23 cc.: No free HCl, residue normal. Contents after test meal, 30 to 50 cc.: no free HCl, total acidity 4 to 6, Pepsin 5 to 7%, rennet positive in undiluted contents only. Streaks of blood (tube). *Stools:* Usually normal, occasionally microscopic excess of meat fibers and connective tissue. Guaiac negative. The patient's general condition improved considerably under rest, diet, tonics and careful hygiene, but the achylia persisted unchanged for five months under observation, at the end of which time the gastric secretion was found to be entirely normal. Free HCl, 25. Total acidity 41. Pepsin 80%. Rennet positive in 1-300 dilution. Several examinations in the last six months show that this normal condition has continued without variation. Headaches and other nervous symptoms persist.

CASE II. Age twenty-seven. Laborer. Family and previous history good. Tea slight excess. Tobacco moderate. No alcohol. For six months gas and distress after meals. Bowels regular. Best weight 160, now 154. Physical examination: Vigorous, young man. Not neurotic. Hemoglobin 95%. Reds 5,400,000. Whites 8,000. *Stomach,* fasting contents, 20 cc.: Clear fluid. No free HCl. Contents after test meal 20 to 50 cc.: No free HCl. Total acidity 2 to 4. Pepsin 0. Rennet positive in undiluted contents only. Stools: Normal, except for excess of microscopic meat fiber and connective tissue. Guaiac negative. General improvement under treatment, gained 5 lbs. Stomach contents have remained the same during the last year.

CASE III. Age twenty-seven. Merchant. Previous history good. Alcohol and tobacco very moderate. Neurotic temperament. For ten months distress after meals. Poor appetite. Belching of gas. Bowels regular. Objective examination: Well developed and nourished. Hemoglobin 90. Reds 5,400,000. Whites 7,400. *Stomach contents,* after test meal, 47 cc.: No free HCl. Total acidity 1. Pepsin 0. Rennin 0. *Stools:* Normal, except for moderate excess of microscopic meat fiber and connective tissue.

Guaiac negative, with meat-free diet. General improvement under treatment, gain of 4 lbs. Several examinations during the last year showed that the achylia remains absolute.

CASE IV. Male. Age thirty-three. Carpenter. Previously well. No bad habits. Two months occasional attack of diarrhea with colic. No gastric symptoms. Headache. Fair appetite. Sleeps well. Neurasthenic. Objective examination: Nutrition good. Healthy appearance. Hemoglobin. 90%. Reds 4,800,000. Whites 7,000. *Stomach contents*, after test breakfast, 30 cc.: No free HCl. Total acidity 5. Pepsin 10%. Rennet positive in undiluted contents, not in dilutions. *Stools:* Watery, foul, with mucus and excess of calcium soaps, and very many well preserved muscle fibers. Guaiac test negative, with meat-free diet. On careful diet and rest, subjective symptoms disappeared. When last seen, had no trouble with stomach or bowels for several months. Gained 5 lbs. The achylia remained almost absolute.

CASE V. Age thirty-six. Shoemaker. No previous illness except "lung trouble," four years ago, from which he entirely recovered. For one year distress several hours after meals. Gas, sour taste, rarely vomits. Fair appetite. Bowels irregular. Very nervous. Best weight three years ago, 180; now 161. Physical examination: Healthy appearance. Hemoglobin 85%. Reds 5,200,000. Whites 7,500. Fasting *stomach* empty, contents after test meal, 30 cc.: No free HCl. Total acidity 2 to 5. Pepsin 5 to 12%. Rennin positive in 1–1 to 1–10 dilutions. Blood streaks. Fragment of gastric mucosa in wash-water was normal. *Stools*: At times practically normal. At other times thin fluid, with macroscopic residue. Mucus, excess of microscopic muscle fibers. Guaiac negative. Under suitable treatment patient became almost entirely well. Stomach contents remained the same during the last three months. Achylia without stasis.

CASE VI. Age forty-three. Merchant. Previous history good, except for considerable alcohol at times. For two months, distress after meals. Afraid of solid

food. No vomiting. Headache. Belching of gas. Constipated at times. Many nervous symptoms. Weight three months ago, 165, now 142. *Physical examination:* Well developed, fairly nourished. Sallow. Poor teeth. Hemoglobin 75%. Reds 4,500,000. Whites 8,000. Fasting *stomach,* no residue obtained. After test breakfast 10 to 50 cc.: No free HCl. Total acidity, 2 to 6. Pepsin 0. Rennin positive in undiluted contents only. Occasional blood streaks. Fragment of gastric mucosa found in wash-water was normal histologically. *Stools:* Pasty. Macroscopic meat at times. Mucus, calcium soaps. Guaiac negative.

Patient slowly and steadily improved; in subsequent ten months, gained 8 lbs., but the above symptoms persisted in milder degree. Frequent examinations demonstrated the persistence of the achylia. A suspicion of early cancer was raised by the age, rapid loss of weight, slight anemia and anacidity, but the course of the disease and the persistent absence of blood from the stools have vindicated the diagnosis of simple achylia.

Case VII. Age seventy-three. Retired coachman. Always well till present illness. No tobacco or alcohol used. Occasional attacks of diarrhea in last few months. Stools 2 to 4 a day for 3 or 4 days. No distress or pain after meals. No vomiting. Appetite good. Feels weak and a little short-winded. Has lost 21 lbs., in the last year. *Physical examination:* Well developed. Fairly nourished. Hemoglobin 70%. Reds 4,500,000. Whites 7,000. *Stomach,* fasting contents 5 cc.: No free HCl. No mucus or food. After test breakfast 10 to 40 cc.: No free HCl. Total acidity, 2 to 5. Pepsin 0. Rennin 0. Fragment of gastric mucosa showed slight granular gastritis. *Stools:* Pasty, foul, mucus, with leucocytes and epithelium, occasionally gross meat and connective tissue residue. Excess of fatty acids. Guaiac negative with meat-free diet. General condition steadily improved under careful diet and iron. Fifteen lbs., weight was gained in ten months. Attacks of diarrhea ceased and patient felt well and returned to light work.

Frequent examinations showed the continuance of absolute achylia. A diagnosis of cancer was made in this case by another physician, but our diagnosis of simple achylia was verified by the course of the illness.

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DISCUSSION.

Dr. John A. Lichty, of Pittsburg: There is one point in regard to which I would differ with Dr. White, and that is, the ease of recognizing achylia gastrica. It is very easy to make the diagnosis of achylia, that is, an absence of gastric juice, but the diagnosis of *achylia gastrica* is not made so readily. Some of these cases follow a very peculiar course. I have in mind a case which Dr. Einhorn recognized as one of achylia gastrica ten or twelve years ago, and when I saw the patient, six years ago, I made the same diagnosis. The patient subsequently developed pernicious anaemia, with the typical blood picture of that disease, and died. I am inclined to doubt now that that was an absolute case of achylia gastrica. The achylia was present beyond doubt, but I think we had the beginning of pernicious anaemia from the outset. Of course, we are all willing to admit that both of these diseases are established on an equally poor clinical basis, and that it is difficult to make an early absolute diagnosis of either, but I believe that some of these cases of so-called achylia gastrica will, if carefully followed up, be found to drift into a fatal pernicious anaemia.

Another condition is the achylia gastrica or absence of gastric juice, but the diagnosis of *achylia gastrica* is not or intense grief. Based upon a gastric analysis, we could diagnose such a case achylia gastrica, but if followed up it would be found that the loss of the gastric secretions was only temporary.

There is another point to which I would like to refer, and that is, the diarrhoea that may accompany an achylia gastrica. Frequently, it is the first symptom that elicits the patient's notice. Such a diarrhoea will persist in spite of the use of the various astringents, while hydrochloric acid will usually have an almost magical effect upon it, curing it within a few days.

Dr. A. L. Benedict, of Buffalo: I agree almost entirely with the reader of the paper, and will limit myself to only a few points brought out by Dr. Lichty. I believe these cases of achylia gastrica can properly be divided into three groups: I. Those connected with cancer of the stomach or chronic gastritis. 2. The permanent cases that are apparently functional, although it seems curious that such a condition should exist without organic disease. 3. Temporary achylia, where we have also a lack of secretion on the part of the kidneys, and perhaps of other glands.

In some of these cases it seems as if we have an illustration of the process of evolution. For instance, in the nut-eating animals salivation is a very active factor in the digestive process, whereas in the human race it has only a faint action on cooked starch. In the herbivorae, the saliva is a much more important factor than in the human race, in whom there seems to be a tendency to depend more largely upon the gastric and intestinal secretions, and to a very slight extent upon the saliva.

I have adopted the following method of estimating the stomach contents in these cases, which I have found very

simple and reliable: Boil the albumin and centrifuge, and by bulk we should get normally two per cent of moist albumin. Then add phosphomolybdic acid, and the additional precipitate, should be from ten to twenty per cent. In cases of achylia gastrica, the first precipitate may be very slight or entirely absent, and the second may be reduced to one to three per cent.

I have never been able to understand why some of these cases are associated with anaemia, and others are not. In some cases of achylia, the administration of hydrochloric acid seems to stimulate the intestinal and pancreatic functions. I have found achylia gastrica and also an achylia of all the digestive glands associated with Addison's disease; in such cases, the food simply passes through the alimentary canal, without undergoing very much change excepting decomposition.

Dr. J. Kaufmann, of New York: I can confirm the statement of Dr. White that achylia gastrica is not an infrequent condition. While I am not prepared to give figures, I have certainly seen more than one hundred cases. I think we could understand the condition better if we did not speak of achylia gastrica as a disease *per se,* but merely as a symptom. It is a functional disturbance, which may be associated with different condition of the stomach. It is easy to understand in all those cases where it is associated with chronic gastric catarrh or cancer, and where the gastric glands are destroyed, but it is more difficult to understand in those cases where it has been shown that the gastric glands are well preserved. Here we have to deal with different groups of cases. It may be that some people were born with this functional defect, just as we see people who are born color blind. I have had occasion to follow up a number of cases for many years—ten years or more—and frequent examination of the stomach secretions during those periods have convinced me that the achylia is permanent. I have never seen the secretion return in a case where the complete lack of secretion had once been demonstrated. In some of these cases, the condition is discovered by mere chance. Under diet, and even without proper diet they are apparently perfectly well. I think it would be interesting to learn whether this condition of a lack of gastric sercetion is not found in infants and young children.

In certain cases, the lack of gastric secretion may be due to disturbance of the nervous system, as Dr. Lichty has reported. In another group of cases, the excretion of toxins by the stomach may be the cause of achylia. We know that the stomach is not only a secretory but also an excretory organ. Morphine, given hypodermically, is excreted by the stomach, and the same is true of other substances, foreign as well as such substances as are formed in the body. For example, urea is excreted by the stomach in uraemia. The excretion of toxins by the stomach may also be the cause of achylia in certain cases of pernicious anaemia. I think it is quite possible that in some instances the achylia gastrica is an effect rather than

a cause of pernicious anaemia. The same toxins that exert a deleterious effect on the constituents of the blood may also have an injurious effect on the gastric mucosa. This same theory had some bearing upon the number of cases of gastric cancer, which showed anatomically that the glands were well preserved, in spite of a complete lack of gastric secretion. I believe there is no doubt that toxins play an important role in cancer of the stomach and other organs. Our present methods of examination of the stomach contents do not help us to distinguish between cases of achylia resulting from disturbances of the nervous system, or from cancer, or atrophic processes, or those due to toxaemia. We must rely on other symptoms to make the differential diagnosis. The development of the clinical picture often arouses the suspicion that cancer may be at the bottom of the trouble. The sudden onset of digestive disturbances, accompanied by a marked impairment in the patient's general condition should always arouse the suspicion of malignant disease, and justify in certain cases a probatory laparotomy.

Dr. White: While in the majority of cases of achylia gastrica the diagnosis is comparatively easy, there is a minority in which it is very difficult indeed. In the simple type of cases I have included those in which there is no severe anaemia or other sign which would indicate another disease to which the gastric condition could be secondary, and in this group the diagnosis is comparatively simple.

Concerning the use of hydrochloric acid in these cases, I think that if it is to be used at all, the doses must be very large in order to get any free acid with the ordinary full diet. The use of such large amounts is often impracticable. In my experience, the improvement which occurred was due more to the diet and hygiene than to the use of drugs. The term achylia gastrica is the best and only appropriate name for the simple primary cases, but in the secondary type we are dealing with a symptom rather than a disease.

TWO CASES OF GASTROSUCCORRHEA OR GASTRIC HYPERSECRETION ASSOCIATED WITH ATROPHIC CIRRHOSIS OF THE LIVER.*

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I WISH to report to you two unusual cases of gastrosuccorrhea or hypersecretion of the stomach. The special points of interest in connection with the cases were the following:

(1) The cases represented, in their special symptomatology, the history of the symptoms and the character of the findings revealed by the examination of the contents of the stomach, typical cases of gastrosuccorrhea, one of continuous hypersecretion, the other of intermittent or periodic hypersecretion; and this stomach symptomatology represented, at the time the cases first came under observation, the prominent and only definite symptoms present.

(2) The special pathological conditions of the stomach, with which hypersecretion such as was present in these cases is ordinarily associated, that is, which most frequently prove to be the primary causes of hypersecretion, gastrectasis, or ulcer of the stomach, were not present in these cases. This fact was proven in the first case, that of continuous hypersecretion, by the findings at autopsy in addition to the results of clinical examination. In the second case it was indicated by the results of clinical examination.

(3) The pathological condition of cirrhosis of

* Read at the annual meeting of the American Gastro-Enterological Association at Boston, June 4, 1906.

the liver was present in both cases. In the first case atrophic cirrhosis of advanced degree was found at autopsy. In the second case the diagnosis of cirrhosis was based upon the finding of an enlarged liver plus the marked alcoholic history of the patient.

As you know, disturbance of the stomach, usually of a general or indefinite character, sometimes including morning vomiting sometimes hematemesis, is a common associate of liver cirrhosis. These two cases are, however, the only instance of which I have a record in which the stomach manifestations presented the definite symptom complex of gastrosuccorrhea, or at least where the existence of such a symptom complex has been determined or recorded. There are cases reported of hyperchlorhydria with some hypersecretion associated with cirrhosis. These cases are, however, cases of hypersecretion, not cases of hyperchlorhydria.

The first case was one of continuous hypersecretion, or Reichmann's disease. The existence of atrophic cirrhosis was determined at autopsy. The record of the case is as follows:

The patient, a male, age fifty-two, was first seen by me in November, 1904. Up to about a year previous to this date the patient had considered himself a well man. At this time, about December, 1903, he began to be troubled with what he called dyspepsia. The only symptoms of this dyspepsia consisted of a sense of discomfort in the stomach coming on three or four hours after the morning meal and lasting more or less all day. There was a sense of fullness and some swelling of the abdomen, with much eructation of gas; no vomiting. The bowels, which had previously been regular, became constipated. Coincident with, or soon after the appearance of, this dyspepsia the patient noted loss of strength, loss of appetite, and a loss of flesh gradually appeared.

From this time on the symptoms of stomach trouble increased in severity. At the start the dyspepsia was intermittent, appearing by spells of a week or two, with periods of comparative freedom of from two to four weeks. As time went on the symptoms became continuous, and severe attacks of pain in the stomach and occasional vomiting occurred. During the last two or three months the pain had been very severe at times, beginning with a dull ache early in the day and increasing until late in the day or in the night, when it became severe and vomiting usually occurred with temporary relief of pain. Of late, vomiting had occurred daily two or three times a day. It occurred more commonly late in the day or at night, but some times came in the morning before he had eaten. Pain and vomiting were not especially connected with the taking of food. He vomited quite as frequently when on a limited diet, and as often four hours after a meal as directly after. Food never relieved symptoms. There was some water brash and regurgitation. The fluid raised was not especially acid as far as the patient could observe, but felt like pure water.

The vomitus consisted of large quantities, often a quart or so of greenish colored, very watery material. In some instances it contained no food, especially when it occurred in the morning. No blood had been noted in the vomitus.

There had been great loss of flesh and strength, shortness of breath on exercise. The secretion of urine had been small. There had never been œdema of the legs.

The past history of the patient contained little data which had any apparent bearing on the present condition, excepting that he had been a moderate drinker for thirty years. He had used tobacco freely. He had never had syphilis.

The record of physical examination was as follows: Patient of a dusky pale color and much emaciated. Hemoglobin, 65%; red corpuscles, 4,900,000; white corpuscles, 4,600; coagulation of blood rapid. Temperature, 98.4°; pulse, 100, small. Lungs negative. Heart normal in size and sounds. Abdomen was rather

full compared with the general emaciation of the body. There was no ascites. Some enlargement of veins of wall. Liver dullness extended from the seventh rib in right nipple line to within a half inch of costal border. The edge could not be felt. The spleen was not palpable, but large to percussion. There were no glands and no tumors to be felt. The arteries were somewhat sclerosed at the wrists. There was no œdema of scrotum or legs. Reflexes were normal. The urine was concentrated and contained the slightest possible trace of albumin and no sugar. The sediment contained a rare hyaline cast, an occasional epithelial cell and red blood corpuscles. The contents of the stomach, expressed by tube in the morning after a fast of twelve hours, showed the following characteristics:

Quantity, 2,000 cc. The character was that of a dirty, greenish colored, watery fluid. A moderate quantity of solid matter, which consisted principally of green flakes of amorphous matter, was present. No food remnants which could be recognized as such by the microscope were present. The quantity of microorganism yeast bacteria was small. The contents had an acid reaction. Free HCl was present. The quantity was 0.03%. No lactic or other organic acid was present. The total acidity was 0.08%. No blood was present by the guaiac test. The fermentation test was negative. Inflation of the stomach showed the lower border about one half inch below the navel. The top border was normal. The stomach appeared as rather a large stomach.

The patient was given a Leube test meal and the contents expressed at the end of eight hours. The character of the contents was as follows:

Quantity, 480 cc. Color greenish; consistency watery, with a moderate sediment. The sediment contained a rare starch granule, a fat globule. Free HCl was present. Total free HCl was 0.08%. Total acidity was 0.18%.

The clinical picture of this case was, as you see from the records of the history and clinical examination, characteristic of the condition of

continuous hypersecretion of the stomach. The emaciation and debility of the patient were extreme, but as you know, this extreme condition is seen in chronic hypersecretion, and, if we omit the tendency to enlargement of the veins of the abdominal wall, there was nothing in the general picture which did not go with the diagnosis indicated by the record of symptoms and physical finding connected with the stomach. The cause of the condition of hypersecretion could not be determined at the time. Of the common causes, stasis and ulcer of the stomach, stasis was excluded by the absence of food remnants in the fasting contents and the character of the contents obtained after the test meal. The large amount of fluid contents is not in itself an evidence of stasis as the autopsy finding in this case proves.

There was no direct evidence of ulcer either in the history or in the physical findings. The low quantity of free hydrochloric acid in the gastric contents did not favor the existence of this condition as a cause of the hypersecretion. There was no hyperchlorhydria such as is sometimes associated with hypersecretion.

The condition is said to occur as a neurosis independently of any discoverable pathological lesion in the stomach or elsewhere in the body. Whether or not we were dealing with such a condition in this case could only be surmised. The extreme emaciation of the case suggested carcinoma of the stomach. I have, however, no record of a marked hypersecretion such as was present here found in association with cancer of the stomach not associated with stasis.

Six weeks later the patient died. The symptoms subsequent to my observation did not differ from those recorded. The autopsy showed atrophic cirrhosis of the liver. The stomach was

disclosed by autopsy findings, and of stasis of any kind, as disclosed by clinical investigation. The conception of a connection between the two conditions in the other direction, the long standing hypersecretion serving as a contributing cause to the cirrhosis, has, as far as I know, no facts of medical observation to support it. It is not suggested by the history of this case.

The second case which I have to report is one of intermittent or periodic hypersecretion. The existence of cirrhosis in this case is based upon the finding of an enlarged liver combined with the alcoholic history of the patient. The patient is still alive. The record of the case is as follows:

The patient, a bartender, age forty-four, was first seen by me in February, 1905. For about a year previous to this time he had been having acute attacks of stomach trouble, alternating with periods of well being. The symptoms of the attacks were as follows: Following a period of well being the patient would be seized with severe pain in the stomach, followed by vomiting. Following this for a period of one to three weeks there would be constant nausea with frequent vomiting, distaste for food, excessive thirst. The vomiting, as a rule, followed the ingestion of food in an hour or so, but some times came in the early morning or long after eating.

The vomitus consisted of large amounts, was watery and acid. It had never contained blood. At the start of the attacks the pain was very acute and relieved temporarily by the vomiting. The pain was at the pit of the stomach and beneath the sternum, never in the right side. There had been no jaundice. The first attack which occurred lasted one week. The patient was then perfectly well for three months, and then had a second attack lasting two weeks. Following this there was a period of well being lasting two months and then the present attack. There was loss of flesh during an attack and a re-establishment in the intervals. The patient had been a steady drinker. No

special occasion for the advent of the attacks could be discovered. He was frequently drunk during the intervals of well being. He denied syphilis. The condition of the patient at the time of my visit was as follows:

Well developed; color, good; hemoglobin, 100%; temperature normal; pulse 80; heart and lungs normal; abdomen full; no ascites; no enlargement of veins of abdominal wall. Liver dullness extended from sixth rib nipple line to two inches below costal border. Edge palpable and symmetrical. Spleen not palpable. Arteries were normal to the touch. Reflexes normal.

The contents of the stomach, expressed in the morning after a twelve hours' fast, had the following characteristics:

Quantity, 500 cc. Character, watery with very little sediment. No food was present. Free HCl present; quantity was 0.12%. Total acidity, .14%. No lactic acid. No blood by guaiac test. Inflation of stomach normal.

The contents expressed, two hours after an Ewald meal, had the following character:

Quantity, 250 cc; Consistency purely liquid. No food was present. Free HCl, 0.22%. Total acidity, .28%.

The attack of stomach disturbance in which I saw the patient lasted three weeks. It was followed by a period of comparative well being. During the latter period the patient suffered from dyspeptic symptoms consisting of distress after eating, eructation of gas. There was no vomiting, and the patient considered himself as well as usual. The contents of the stomach obtained during this period expressed one hour after an Ewald meal, showed the following character:

Quantity, 150 cc. The proportion of supernatant fluid was rather greater than normal. Total free HCl was .16%.

Three months from the time of my first visit the patient had an acute attack similar to the one described lasting two weeks. Since then he has had two similar attacks. The size of the liver remains practically the same.

We have here a case of periodic or intermittent hypersecretion associated with liver cirrhosis. The cause of the attacks cannot be definitely determined. They may be due to accute irritation of the stomach from indulgence in alcohol. Local stomach causes, as stasis or ulcer of the stomach, can apparently be ruled out by a consideration of the results of clinical investigation. How far the cirrhosis is a contributing cause is a matter of surmise. The case, however, serves as a second example of the association of cirrhosis and gastric hypersecretion.

DISCUSSION.

Dr. Max Einhorn: I would like to mention one case which was not embodied in my paper this morning, but which seems to belong to this class. There was a history of alcoholism, as in Dr. Hewes' case, with severe gastric symptoms extending over a period of fifteen years. Food taken would usually be promptly vomited. When I first saw the patient, he had lost about 50 or 60 pounds. Upon examination I found a much-dilated stomach, with symptoms of stagnation of food, hypersecretion and hyperchlorhydria, together with cirrhosis of the liver. His stomach, after lavage in the evening, would fill up with gastric juice during the night, to the extent of a pint and sometimes even a quart. There was continuous hypersecretion, independent of the stenosis. The patient was treated in the New York Post-Graduate Hospital, and when it had been demonstrated that even liquid foods would not pass through the stomach, it was decided to do a gastro-enterostomy, although the cirrhosis of the liver rendered the prognosis unfavorable. At that time there were also evidences of slight gastric hæmorrhages. The abdomen was opened, and the cirrhosis of the liver was verified at the time of operation. There was also a stricture of the pylorus. The patient died a week later.

In this case there was apparently a hyperchlorhydria, which led to the development of an ulcer, and the stenosis probably resulted from the close proximity of the ulcer to the pylorus. This case seems to belong to the same type as those reported by Dr. Hewes.

Dr. J. Kaufmann: Since this question of gastro-succorrhoea and stenosis has come up for discussion, I wish to add a few words. The very fact that such large quantities of fluid were found in the fasting stomach in the cases reported shows that there must have been stagnation. There is no reason why this fluid should not have been removed into the intestine, unless there was some impediment. In most cass that have been described as true gastro-succorrhoea, the anatomical findings were always those described by Dr. Einhorn, including the presence of stricture. If, after washing the stomach in the evening, you find such a large quantity of fluid in the stomach the next morning, that certainly indicates that the emptying of the fluid into the intestine was prevented by an organic or spastic constriction. An examination will reveal small quantities of starch in the stagnated stomach contents.

Dr. Hewes (closing the discussion): In reply to Dr. Little's question I would say, that the duodenum was examined and found intact. Dr. Kaufmann states that the presence of so large a quantity of fluid in the stomach is an evidence of stasis. I should say that the records of this case directly disproved this contention. There was

no obstruction of the pylorus present by autopsy findings, and by clinical findings the stomach emptied itself of food within the normal period. Stasis was certainly not a primary cause of the accumulation of fluid or the hypersecretion in this case. Whether or not the hypersecretion may be a cause of spastic stasis is not the question. Starch granules in small number may be found in the fasting contents, after a twelve-hour fast in normal individuals.

Extracted from the American Journal of the Medical Sciences, May, 1907

THE RELATION OF THE KIDNEYS TO GASTRO-ENTEROLOGY.

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In a broad way, renal diseases may be classified as follows: (1) Neoplasms, of interest mainly to surgeons and pathologists. (2) Local pathological lesions, such as infarcts, scarcely diagnosticable. (3) Parasitic disease: (*a*) gross animal parisitism, rare in this climate; and (*b*) bacterial infections, septic, gonococcic (ascending), tuberculous, exanthematous (included as a matter of convenience), and other acute infections. (4) Traumatisms. (5) Necrosis. (6) Displacements. (7) Calculus disease and certain forms of urinary obstruction. (8) Inflammations or degenerations, involving the kidneys generally (Bright's disease, etc.). (9) Functional disturbances: (*a*) motor, impossible, except in an indirect sense of involvement of the vessels in ischemia and congestion; (*b*) secretory, excessive, deficient, or perverted; and (*c*) sensory, neuralgia (also, true neuritis of the kidney).

While almost any kind of disease of any part of the body may occasionally produce, or be accompanied by, some definite disturbance of digestion or call for special attention to diet and metabolism, the following partial classification of renal conditions is suggested as pertinent to our consideration: (1) Mechanical influences: (*a*) movable kidney, and (*b*) calculus disease and urinary obstruction. (2) Chemical and allied influences: (*a*) acute nephritis; (*b*) chronic nephritis; (*c*) renal paresis and paralysis; (*d*) polyuria; and (*e*) urinary abnormalities secondary to digestive and metabolic disturbances, without strict renal disease.

The relatively large number of cases of movable kidney encountered in digestive practice must be significant. Even gynecological series do not usually show more than 10 per cent. Ralph C. Larrabee[1] found 112 cases among 272 women—41.5 per cent. Wolkow and Delitzin[2] found 46 per cent. Others have reported an incidence of 60 to 80 per cent. The personal equation must enter into the matter. I have considered as movable only those cases which

[1] Boston Med. and Surg. Jour., November 26, 1903.

[2] Die Wanderniere, 1899.

showed an unmistakable, palpable change of position beyond anything that could be ascribed to elasticity of the abdominal wall. In my cases no significant association with any particular digestive disease has been demonstrable, certainly not with inflammation of the appendix, nor with genuine gastroptosis. I have found altogether, in many hundred cases, only two of hepatoptosis, and have not been convinced of a typical general splanchnoptosis. But all of my results, in any line of investigation, are counted as negative unless unmistakable abnormality has been found on rigid objective study. If one counts every case of atonic dilatation of the stomach as gastroptosis, every case of flabby abdomen as enteroptosis, and the combination of two or three ptoses as a general splanchnoptosis, it is easy to make up comparatively large series. It should be distinctly understood that this statement is made without prejudice and without the least idea of reflecting on the much larger series of several competent observers, abroad and in America. Differences of clientele may easily affect the statistics.

STATISTICS OF MOVABLE KIDNEY FROM RECENT PRIVATE PRACTICE, NOT INCLUDING THE PRESENT FISCAL YEAR.[3]

	No. in series.	No. of cases.	Percentage of incidence.
Males living	321	15	4+ per cent.
Females living	314	83	26+ "
Total living	635	98	15+ "
Males dead	120	4	3+ "
Females dead	82	9	11— "
Total dead	202	13	6+ "
Total males	441	19	4+ "
Total females	396	92	23+ "
Total patients	837	111	13+ "

ETIOLOGY. I have found very few cases of movable kidney among two or three hundred children from early infancy to puberty, and, so far as my experience goes, the following classic causes are not operative: loss of weight, frequent or even any pregnancy, corsets, heavy lifting, straining, traumatisms, exhausting sicknesses, etc. I have made out no significant hereditary or familial tendency, although it is common enough to find it in two cases in a family, in the same or different generations. It is not significantly associated with hernia and other displacements of viscera, although such coincidences are, of course, quite frequent. The essential cause seems to be failure of abdominal development at puberty, when the pelvis enlarges.

[3] Not counting series examined in asylums and hospitals especially to find movable kidneys, I have careful records of 170 cases of this condition, the recognition of which in this country has been comparatively recent. A larger series, from the records of Dr. C. D. Aaron, will be discussed with these in a subsequent paper.

Nephropexy often fails signally and there is, perhaps, danger in cutting, even with needle wounds, excretory tubules. Curiously enough, I have found movable kidney especially frequent in physicians' wives. In many cases the gastric symptoms which lead the patient to consult a digestive specialist seem purely reflex, the physical and chemical examination of the stomach resulting negatively. None of my nearly 300 cases has had Dietl's crises. One was supposed to have had but a careful examination and inquiry into the history failed to support the diagnosis. Without questioning the authenticity of reports of this condition, it certainly must be extremely rare, and one is tempted to suggest that surgeons may make the diagnosis without adequate ground, and to draw a parallel with the unprecedented frequency of gastric ulcer in surgical series, as contrasted with medical and postmortem statistics.

Renal Calculi and Urinary Obstruction. These conditions, though often marked by vomiting and constipation, the latter even amounting to obstipation and due to reflex spasm, do not directly come under our observation for acute attacks. The prophylaxis of calculus disease in general, however, is a very important function of the digestive specialist. Routine examinations of the urine in patients subject to urinary calculi should always include an investigation of the acidity, a matter which will be discussed in another article.

The differential diagnosis of renal calculus is sometimes exceedingly difficult. Other forms of obstruction, biliary lithiasis or inflammation, scolecitis (appendicitis), colic of the appendix, inflammatory conditions of the bowel, especially limited as to the ceco-appendix, sigmoid, etc., gastric ulcer, pancreatic disease, and, in cases of suspected hydronephrosis or pyonephrosis, any tumor, splenic or otherwise, may be confused.

J. B. G. (No. 56, of 1904–05) had an acutely painful attack which his attendant considered biliary colic. An excellent general practitioner, called in consultation, unhesitatingly diagnosed renal colic, although, in justice to him, it should be said that he had somehow obtained a false idea of certain conditions, including hematuria, which had never been actually demonstrated. The patient was subsequently turned over to me, there being some fever and leukocytosis, but only average urinary acidity. My diagnosis lay between biliary lithiasis and some septic involvement of the appendix. Operation was refused. Some months later another attack led to operation and a gangrenous appendix was found. Death followed. In other cases it has been found impossible to distinguish between renal colic and sigmoiditis or left-sided colitis, at least without following the case past the acute attack.

Nephritis. Acute nephritis occurs mainly in connection with infectious fevers or such exposure to chill and metabolic or ordinary saprophytic toxins as withdraws it from our experience. In 713

serial cases I saw it only once in consultation. Nephritis or even marked congestion does not occur so frequently in typhoid as in the fevers outside of our specialty, and, if typhoid patients are adequately fed, on cereals as well as milk and beef-juice, and some pains are taken to prevent intestinal decomposition, renal complications will be exceedingly rare.

Chronic nephritis is quite infrequent in digestive practice, especially when we consider the frequency of hepatic sclerosis and the history of alcoholic indulgence, drugging with arsenic and various other irritants, and the formation of toxic products in the alimentary canal or in general metabolic processes.

Among 517 living patients, 4 showed chronic diffuse nephritis, 1 multiple visceral lesion (A. O., M. R., T. R., anemia, chronic nephritis, some cerebral lesion producing temporary aphasia, apparent recovery reported by attendant), 2 persistent albuminuria without casts (one of the last, orthostatic albuminuria). Among 196 dead subjects, 8 showed chronic or subacute tubular nephritis, the diagnosis as to the dividing line being difficult in such cases, which are under observation for a short time previous to death; 12 showed multiple visceral lesion, although in some there was merely albuminuria and no casts, so that the kidney may be considered as only congested; 1 showed albumin and albumose, the nature of the case being puzzling (P. S., No. 60, 1898–9); 1 showed renal insufficiency and auto-intoxication without genuine Bright's disease. Altogether 713 cases included only 29 that could be considered as distinctly renal lesions, an incidence of 4 per cent. for the whole series, about 1.5 per cent. for the surviving cases and 11 per cent. for the fatal cases. On the other hand there were several of extreme cardiac disease, some with hepatic sclerosis of marked type, atrophic or hypertrophic, abdominal or general dropsy and ultimately pulmonary œdema in which the kidneys maintained themselves to the end. In one case of multiple visceral lesion there was a terminal bronchopneumonia, general dropsy, and a decided right-sided anasarca appeared a few days before death. The pneumonic condition was more marked in the right lung, and there was found postmortem more fluid in the right pleural cavity, but the sharp unilaterality of the dropsy could not be explained by posture, nor by anything observed before death or at the necropsy. It was considered to be due to a unilateral angioparalysis, but this only makes the ultimate explanation more difficult.

With reference to the diagnosis of renal lesions from the urine, allusion may be made to the classification of urinary casts by Simon and others, to a recent series of articles by Emerson, and to Cabot's valuable skeptic comparison of clinical and postmortem results, published last year. The conflict between the older and some of the more modern views may be harmonized to some degree, if we regard the matter with the following practical qualifications:

1. Minor lesions of the kidneys, demonstrable by microscopic examination of sections, may escape recognition on account of the dilution of diseased by healthy urine.

2. On the other hand, foci of inflammation or degeneration may produce albuminuria, casts, etc., and ultimately recover or be filled in by scar tissue.

3. Even quite extensively diseased kidneys may occasionally excrete urine that eludes an ordinary clinical search for abnormalities.

4. Normal urine contains a trace of proteid, and any normal organ must shed more or less epithelium or be subject to conditions practically inflammatory, but of minute area.

5. Under strain any kidney will pass albumin and casts. Whether the condition should technically be considered nephritis or not is not of practical importance.

6. Unless foreign albumin is introduced directly into the circulation or is absorbed from excessive amounts ingested, as after a diet of raw eggs, there is no condition analogous to hyperglycemia. Hence there can be no such thing as p hysiological albuminuria, excepting as stated. It is exceedingly doubtful whether an albuminuria can be functional.

7. On the other hand, unless an albuminuria has an obvious source below the kidney, casts are rarely lacking, and a clinical albuminuria, not readily attributable to the lower passages, is almost always an indication of true renal congestion or nephritis.

8. Unilateral renal disease without obvious cause and indications is rare.

9. For clinical purposes albuminuria can be conveniently estimated by boiling and centrifuging, the moist precipitate exaggerating the actual percentage of albumin five or six times. With due allowance for temporary fluctuations, bulk of urine, and the particular kind of renal lesion present, the amount of albumin is an approximate measure of the grade of the lesion, although it would be obviously absurd to draw prognoses directly from the centrifuge readings.

10. The ordinary clinical observer (a personal confession is here included) cannot always make a sharp classification of the different kinds of casts nor always distinguish between true and false casts. Whether experts can do so or not need not be discussed. However, all studies, in which the attempt has been made to identify every cast seen and to make a diagnosis from the urine alone, at one or a few examinations covering a brief period, show that it is impossible to connect a microscopic field with a definite pathological lesion.

11. However, by considering both clinical, chemical, and microscopic examinations, and by taking the general conspectus of the microscopic examinations covering a reasonable period, I believe that it is possible to distinguish pretty sharply renal congestion, interstitial and tubular nephritis, and, of course, amyloid disease,

hemorrhage, etc., and to gain a fair idea as to the pathological stage of the lesion.

12. Nitrometric estimations of urea are really rough tests for total non-proteid nitrogen. They certainly include uric acid and ammonium salts.

13. The various quantitative tests for urea, ammonia (by liberating ammonia gas and titrating a standard acid solution used to absorb it), uric acid (by R. Harvey Cooke's test), and salines (by titration or centrifuge) are of considerable value in keeping track of metabolism, although they do not yield diagnostic information in the sense of enabling the application of names to renal conditions.

It would require too much space to consider the ways in which renal and strictly alimentary conditions react upon one another. Croftan has emphasized a point not altogether novel, that uremia is not due in a direct, crude sense to the retention of urinary waste. Bouchard's statements that urinary waste kills if accumulated for twenty-four hours, hepatic waste in nine hours, are true only in an illustrative sense. It is doubtful whether, for practical clinical purposes, we can distinguish these sharply. At any rate, their production certainly falls as does the elimination, although not *pari passu.* So, too, in chronic disease of the emunctories, we must assume an increased tolerance to the poisons.

J. J. S. (No. 99, of 1905–06) has amblyopia, diagnosticated as due to albuminuric retinitis. He also manifests the general toxemia conected with hepatic sclerosis and chronic diffuse nephritis, yet forn long periods his urine is qualitatively normal. It has never yielded as much as 1 per cent. of moist precipitate and the elimination of urea, uric acid, and salines is within normal limits. It is scarcely necessary to state that the real toxic matter has not been isolated nor that, if there is any one characteristic toxin, it must be something present in very small amounts.

H. W. G. (No. 1050, of 1895–96) showed mitral regurgitation, albumin, and casts for several years, and the case was considered one of true chronic diffuse nephritis. In 1899, during an attack of cholera morbus, he was anuric for nearly a week, without developing uremic symptoms. Indeed, there is a paradox of pretty general applicability, that anuria, even existing for a week or two, does not produce uremia. In 1900 the patient had a severe attack of phlegmonous erysipelas and was thought to be dying. Since then several urinary examinations, at long intervals, have failed to show any urinary abnormality. Did the erysipelatous toxemia cure the nephritis?

Renal albuminuria without casts is a subject that has occupied much space in the literature of the last few years. Objection has already been made to the terms physiological and functional. Cyclic is also an objectionable term, as in the case of cyclic vomiting. The act that a condition appears and disappears, even at fairly regular

intervals, does not warrant the term cyclic, unless there is a definite and regular dependence upon some inevitably and uniformly recurring circumstance. In one of my cases the albuminuria was distinctly orthostatic, depending upon the vocation of conductor on a street car, the urine passed after rest in bed being free from albumin. Most of my cases have occurred after adolescence. As casts are occasionally found in normal urine, especially after strain and when large volumes are centrifuged, an occasional cast does not indicate a separation of the case from this clinical group. Orville A. Kennedy[4] reports a case at first showing only cylindroids, but after a few years genuine tube casts, and the general consensus of opinion seems to be that these forms of albuminuria are essentially nephritic, though the lesion is mild and even curable.

A very practical consideration is the extent to which the stomach in nephritis acts vicariously for the kidney. We have probably all of us laughed at physicians who have called the jejune vomitus in Bright's disease vicarious urine. Doubtless the expression has been due to an entirely superficial appearance. Yet we must remember that such liquid necessarily contains the urinary salts and certain organic compounds and even notable quantities of urea. At any rate, lavage seems to have not only a local effect, but really to aid in the elimination of toxins.

Delirium Tremens. This symptom-complex is mentioned in this connection because cases of auto-intoxication with sclerotic livers and badly congested or diseased kidneys have so frequently presented delirium that, from the standpoint of the general practitioner, would be properly called delirium tremens. Indeed, the difference of phraseology may be considered as a divergence of diagnosis. It has long since been pointed out that delirium tremens is not drunkenness and that alcohol often relieves the condition. Without any considerable opportunity to study ordinary cases of delirium tremens, I would like to raise the question whether there is anything essential in the symptom-complex that cannot be caused in total abstainers, and whether, considered as a group, all cases of so-called delirium tremens cannot be reduced to auto-intoxication with waste matters commonly eliminated by the liver or kidney or both. No one disease of either organ can be considered necessary. One may seek and often find hepatic sclerosis and chronic nephritis. But, as the late H. F. Formad pointed out, the really characteristic drunkard's kidney is the pig-backed kidney with chronic congestion. Then, too, delirium tremens may occur before hepatic sclerosis has had time to develop.

One of my latest fatal cases (No. 10, of 1896–97), aged forty-three years, was the son of a man who died of typical atrophic hepatic sclerosis. Both had been rather liberal users of alcoholics, though

[4] Amer. Jour. Med. Sci., March, 1906.

the father presented no true alcoholism. The son had had several alcoholic attacks, including such lesions as pericarditis and lobular pneumonia and his liver was quite contracted. In the last attack it was much enlarged, but speedily contracted to its permanent minimum under calomel. He also had right lobar pneumonia. The urine could not be saved until just before death, when it was found to contain 7 per cent. of sugar, but neither albumin nor casts. There had been no previous symptoms of diabetes and the urinary excretion, though liberal, so far as could be judged, was not excessive and there was no polydipsia. Still, so large a percentage of sugar, after several days of very light diet, almost justifies the term diabetes. Necropsy could not be obtained.

Curiously enough, in about fifty cases of delirium tremens, I have never noted hallucinations of snakes; rats, horses, dogs, cats, and human beings have been the most frequent hallucinations. The last patient mentioned saw playing cards on the ceiling. Sometimes the hallucinations have been general, sometimes individual. With rats, lions, tigers, etc., the patient would simply see such animals. Human beings are usually individualized. Domestic animals are sometimes seen as individuals, sometimes merely as animals. In one case, the patient, himself an Irishman, described his hallucinations as of red-headed Irishmen, but apparently recognized none as individuals. Fear may or may not be present and the fear may be a logical realization of danger, as in the case of lions and tigers, savage horses and dogs, or a morbid horror as in the case of rats and mice. Most human beings have more or less ophiophobia, so that if snakes are seen at all there is likely to be mental terror. As a rule, however, terror seems to be lacking and the visions may even be amusing or pleasurable. There is even a story about a zoölogist who experienced scientific pleasure in analyzing the snakes that he saw. With the popular and even professional conception that delirium tremens patients see snakes it is a wonder that suggestion does not lead to the much more frequent presence of this hallucination.

According to one theory, the vision of snakes is not an hallucination, but an illusion due to the congestion of the tortuous and snake-like retinal vessels. I will leave to a more expert physiologist the explanation of how even a congestion of these vessels could lead to sensory impressions of objects of similar contour. That the explanation does not hold is evident from the actual rarity of ophioscopia and from the fact that pasting paper over holes through which the patient saw the snakes enter, or driving them out of the room, or otherwise humoring the patient's delusions, sometimes causes the hallucination to vanish. A much more rational objective explanation is that the blood corpuscles, whose shadows can actually and easily be seen by anyone, form the basis of a visual illusion.

As in other manifestations of auto-intoxication, the prime points in

the treatment of delirium tremens are to stimulate, with digitalis, strychnine, hot saline rectal injections, external heat, etc., to purge and stimulate the kidneys and skin, and to avoid an overdose of any depressant by using a succession of nerve sedatives, rather than any one to excess. Morphine should be used only with the utmost caution.

MULTIPLE VISCERAL LESION is a term that I introduced several years ago, to cover cases in which the heart, lungs, kidneys, liver, spleen, and alimentary canal are mutually involved in chemical and mechanical processes, through the circulation. Sometimes the heart, sometimes the kidneys, sometimes the liver, seems to be the primary locus, but various vicious circles become established. Excepting as dyspeptic conditions or the ingesta directly affect the liver, the alimentary canal is not primarily involved, but eventually becomes catarrhal on account of portal obstruction, and there may be capillary oozing, or the formation of varicose internal ulcers, or massive hemorrhages, due to this interference with return circulation. Pick's frosted liver may exist in such conditions. The pulmonary condition is usually terminal, being a combination of catarrh and œdema.

A very important, and, to me, inexplicable fact is that in renal disease there may be a pretty definite tendency to water-logging with mechanical crippling of the circulation and respiration, or a toxemic state with little or no dropsy. Of course, we expect dropsy in certain disorders of the blood and heart and in tubular rather than in interstitial renal inflammation or degeneration. Then, too, dropsy tends to withdraw soluble toxins from the circulation. But if dropsical effusions prevent toxemia, why are not the same poisons eliminated when the kidneys excrete freely? And why, without any definite line corresponding to the renal condition, do certain patients die of water-logging, without any marked nervous manifestations, while others, without more than a trace of œdema, have convulsions, and still others coma?

RENAL PARESIS is very frequent in disease of the digestive organs. Without any definite renal lesion the kidneys are more or less transiently deficient in function, even allowing for the diet in the particular case. In asthmatic and other patients, we may note an almost absolute paralysis of gastric and renal function for many hours. Food is vomited unchanged and the gastric filtrate has no acid, unless a little due to fermentation; there is no peptone ring and no curdling of milk or digestion of egg albumin. Meantime, no urine is formed. In Addison's disease, we may note gastric achylia, pancreatic and intestinal achylia, so far as may be demonstrated by our present rather crude methods, and the renal function may be much below par. In seasickness, there is a purely functional quiescence along the same lines.

RENAL SUPERSECRETION, commonly termed diabetes insipidus, is a matter that deserves careful scientific study. Apparently diabetes

changes, so that I am confident that the gastric symptoms are attributable to a movable kidney.

In regard to operative measures for the relief of movable kidney, I am convinced that they should always be regarded with extreme caution. When you inflict a wound upon an excretory tubule, whether it is one of large size like the ureter or bile duct, or of microscopic size like those in the kidney, it is possible that you are doing irreparable damage. I recall the case of a woman in whom a third degree movable kidney was found after she had been thrown from a buggy. The kidney was anchored and a rubber tube placed around it as an additional safeguard. Since the operation, the urine has contained albumin and casts, and I cannot help thinking that some of the excretory tubules of the kidney were damaged, and that the operation converted a case of simple movable kidney into one of nephrits of some form.

In reply to Dr. Lichty's question as to why these cases of movable kidney occurred, I can only say they occur because they do occur. I am reminded of the story of the old lady who asked the doctor why some persons were born deaf mutes. He replied, "Madam, it is due to the fact that they came into the world without the power of speech." "Laws," the old lady replied, "My husband says it is just because they were born so. The conditon of movable kidney is met with particularly in the fat and flabby type, and again in the slender, slender-waisted type, with large hips. In many women, in spite of their appearance, the kidney is found securely anchored in its normal position. The subject of the causation of this condition is a very complicated one, and I am convinced that the usual causes to which it has been ascribed, such as tight lacing, etc., have very little to do with it. I look upon it as a developmental effect. In several of my cases, the patients were born prematurely, although it is very difficult to gather statistics along that line.

Reprinted from the New York Medical Journal for October 20, 1906.

THE GASTROINTESTINAL DISTURBANCES ASSOCIATED WITH MIGRAINE.*

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In a paper read before this association at Washington in May, 1903 (*American Medicine*, November 14, 1903), I reported seventy-three cases of migraine studied with reference to the associated digestive disturbances. The conclusions drawn from that paper were, in the main, as follows:

First.—A large majority (sixty out of seventy) of patients afflicted with migraine have some disturbances of the digestive system apart from the disturbances associated with the migrainous paroxysm.

Second.—While most or nearly all patients refer to an excessive acidity of the stomach during or even between the attacks, the majority (twenty out of thirty-one whose stomach contents were examined) had a subacidity.

Third.—Ptosis of one, or more, or all of the abdominal organs is frequently present (thirty-eight out of seventy-three).

Fourth.—A rational consideration of the digestive disturbances in migraine is of some advantage in treating these patients.

Since May, 1903, I have been able to add one hundred and eight cases of migraine to my list, and I am now ready to give the deductions and results from my study and treatment of these cases.

I wish again to refer briefly to the definition of

* Read before the American Gastroenterological Association, at Boston, June 4 and 5, 1906.

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migraine, hemicrania, paroxysmal headaches, or the commonly so called "sick headache." It is a periodical neurosis occurring most frequently in women, and is characterized by paroxysmal attacks of severe pain, usually confined to one side of the head, chiefly in the course of the fifth nerve. At times it is preceded by a variety of transient visual disturbances and paræsthesiæ, and followed by nausea and vomiting (Leszynsky). The headache is often preceded a day or so by an unusual feeling of well being. This affords the patient an incentive for a day of unusual activity. On the following morning she may awake with prodromal symptoms with which she is quite familiar. There may be visual disturbances, photophobia, hemianopsia, blurring of vision, zig-zag shapes of light, or there may be pain and stiffness in certain groups of muscles of the neck or shoulders. These prodromal symptoms often are not present, but nearly every patient has some certain sign which assures her that a headache is due. During the height of the headache light increases the pain, as does also the recumbent position. The characteristic position of these patients is sitting propped up in bed in a darkened room, with eyes closed. Nausea is an early symptom, and vomiting usually heralds the approaching sleep, which ends the attack. Both nausea and vomiting may be absent.

It is decidedly an inherited disease. Of the one hundred and eighty-one cases tabulated, one hundred and sixteen gave a definite hereditary history, while of the remaining sixty-five some gave a doubtful history, and less no hereditary history at all.

It is interesting to note the peculiar type of family to which the migrainous patients belong. Thev are plainly neurotic: chorea, epilepsy, neurasthenia, hysteria, and other neuroses abound. They are small eaters, are below the average weight, have an abundance of nervous energy, but are deficient in

physical strength and endurance. In a crisis they put forth a surprising amount of physical and mental effort, but always collapse with an attack of migraine. Ptosis of one or more of the abdominal organs is frequently found. These families fortunately show the evidences of decay, since a large proportion do not marry, those who do marry often have no children, or have below the average number. Of the ninety-six females in my last list, the age ranged from nineteen to sixty-five years, the average age being 36.2 years, and yet only fifty-two were married and only twenty-four of these had children. There are, of course, some remarkable exceptions. One patient, with terrific headaches, at the age of thirty has had seven children.

Migraine occurs more frequently than is generally supposed. Many cases are overlooked, because the patient in giving her history may refer to attacks of neuralgia or biliousness in such a matter of fact way that no further inquiry is made. A few well directed questions may reveal a trail of sick headaches leading back through two or three generations. The one hundred and eighty-one cases I have tabulated have been selected from slightly over six thousand patients examined—about three per cent. of all patients seen. These figures are all the more striking when the gravity of the disease is fully appreciated.

Migraine usually begins with the establishment of the menses or soon after, and may end with the menopause or soon after; a period of twenty-five to thirty years, which includes the years in which a woman is in her prime. The attacks come every week or two, or every month, and sometimes twice a week. They last from one to two or three days. During the attacks the patient is entirely incapacitated. The number of engagements cancelled, the amount of work left undone on account of sick headache is scarcely appreciated. When one adds to this

the additional fact that very little has been accomplished thus far in overcoming or modifying the attacks, the importance of a consideration of migraine from any reasonable standpoint cannot be overestimated.

It is scarcely profitable to discuss in detail the several theories which have been advanced to explain the cause and symptoms of migraine. The vasomotor theory, the purely nervous theory, the uric acid theory, and the autointoxication theory have all had their advocates and times of ascendence. Some contend that there must be a persistent pathological condition to account for such a decided phenomenon. I agree with those who hold that migraine is of purely nervous origin, and that the symptoms are due to a primary functional disturbance of the nerves themselves. Some of the disturbances, such as vasomotor, may be the result rather than the cause of migraine; others, such as uric acid, autointoxication, digestive, ocular, nasopharyngeal, and menstrual are simply coincidental conditions. Either condition may precipitate an attack of migraine, and thus become an important factor in a case.

In this paper I wish again to call particular attention to the presence and influence of the digestive disturbances. Before doing this I shall give the summary of the cases I have studied and tabulated.

Sex.—Of the one hundred and eighty-one patients, one hundred and fifty-five were females and twenty-six males.

Heredity.—One hundred and sixteen gave a distinct hereditary history. This figure is lower than the actual condition indicates, because many patients were unable to give a satisfactory history, their parents having died when they were in their infancy. In nearly all cases the headaches seem to have been inherited from the mother's side of the family. Occasionally both parents were afflicted, and in such

case the patient usually suffered from a most severe and obstinate type of the disease. Rarely the father alone was afflicted.

Ptosis.—Ninety had prolapse of one or more of the abdominal organs. Most frequently it was the right kidney; next in order, the right kidney and the stomach; occasionally both kidneys and the stomach, and in a few cases all of the abdominal organs. That half of the cases of migraine have ptosis is of some significance and not a mere coincidence. It is another characteristic which marks the type of family in which migraine prevails. From the fact that patients suffering from migraine are almost without exception underfed and under weight it might be concluded that the large percentage of cases of ptosis is a direct result of the disease. The ætiology of ptosis is still obscure, and opinions differ.

Digestive Disturbances.—One hundred and thirty-seven cases gave a definite history of digestive disturbances between the attacks. The symptoms were definite, such as heaviness after eating, pyrosis, fermentation, and gas, constipation or diarrhœa; in fact, any or all of the symptoms which make the symptom complex of what is ordinarily called indigestion. In one hundred and one of these cases gastric analyses were made. Twenty-five had normal secretion of gastric juice, thirty-two had an excessive secretion (hyperacid) and forty-four had deficient secretion (hypoacid). Many showed the evidences of chronic gastritis and enteritis. A few gave the history and had the clinical evidences of chronic ulceration of the stomach.

I stated in my former paper that the opinion usually prevails with both patient and physician that these cases suffer almost without exception from a hyperacid stomach. This opinion is based upon the patient's own experience and statement that what she vomits during the attack is excessively sour. That the stomach contents in the large ma-

jority of cases is not excessively acid is shown by my statistics. In my first series, however, I found a larger proportion of subacidities than in my last series:

First Series.—Thirty-one gastric analyses; eight hyperacid, twenty hypoacid, and three normal.

Second Series.—Seventy gastric analyses; twenty-four hyperacid, twenty-four hypoacid, and twenty-two normal.

Migraine patients sooner or later place themselves on a very strict diet, frequently on a starvation diet. They discard certain foods because of a supposed hyperacidity. They are encouraged in this by their physicians, who too often accept the patient's statement without any investigation. The diet becomes monotonous and the patient is continually in a state of subnutrition, which favors more frequent attacks of headache. It is the recognition and correction of this mistake which has given me great encouragement in the treatment of these cases.

With our very limited knowledge of the true cause of migraine, we cannot look for a specific treatment. But a great deal can be done for these unfortunate sufferers by a rational symptomatic treatment. Such coincidental abnormal conditions as may be found in the eyes, nose, and pharynx, heart, kidneys, digestive organs, pelvic organs, and, in fact, in any organ, should, if possible, be corrected. It is often surprising to see how the slightest abnormality in any of these organs will affect the number and character of the headaches.

In correcting these abnormities, very satisfactory results are often experienced. Especially is this true of the digestive disturbances. Since many of these patients are underfed and upon an unnecessarily strict diet, I have accomplished much in "leveling up" the digestive organs to a higher plane of endurance. The patient thus gains in weight, becomes stronger, physically, does not experience

fatigue so readily, and avoids many attacks. To do this "leveling up" process it is, of course, necessary to be thoroughly acquainted with the condition of the digestive system. This is as important as it is to know the errors of refraction or the muscular difficulties of the eye, before any correction is attempted with lenses.

It is not necessary to go into the details of the various lines of treatment which may be established. I have for some years abandoned all so called specific treatment of migraine. There are two remedies, however, which I have used almost routinely when the removal of reflex and exciting causes was impossible or did not accomplish the desired results. They are the bromides and the various preparations of cannabis indica.

It is impossible in such a series of cases of migraine as this paper is based upon, to give an accurate idea of the results. It cannot be expressed in definite percentages of cures and failures. All that I can say is that many patients experienced relief in the frequency and severity of the attacks, and some seem to have recovered entirely. Of this last class I wish to report one case.

Miss E. M. P., æt. twenty-three years, a stenographer, consulted me first June 21, 1904. She complained of sick headaches, which occurred every third week. The attacks were typical. Her mother and a sister were similarly afflicted. The digestive symptoms between the attacks were, a variable appetite, pain and distress about fifteen minutes after eating, pyrosis and constipation. The patient had been in a street car accident some time previously, and since that time the attacks were more frequent and severe. She had never had her eyes examined. The tongue was coated; the heart and lungs were normal; the stomach was dilated to the umbilicus; the right kidney was movable; the urine was normal; the gastric contents showed a total acidity of seventy and the presence of free hydrochloric acid. She weighed twelve pounds less than her usual weight.

I prescribed a suitable diet and an alkaline treatment. In three months the attacks gradually disappeared.

April 30, 1906. The patient seems well, no headaches of any consequence. The bowels are regular. The stomach is normal, and the right kidney is not palpable.

4634 FIFTH AVENUE.

Reprint from

SURGERY, GYNECOLOGY AND OBSTETRICS

October, 1906, pages 495-501

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APPENDICITIS LARVATA[1]

By ALFRED D. KOHN, M. D., Chicago

EVERY specialist in gastro-intestinal diseases is confronted with a large number of seemingly intractable cases of stomach or bowel disease which refuse to yield to every known method of medical treatment. These cases do not present the classic symptoms of appendicitis; there is almost never any recollection of an attack which is referred to the appendix; but the patients drift on from doctor to sanitarium, and from sanitarium to quack, and from quack to the ever-open arms of the Christian scientist. These patients, after having been unsuccessfully treated by every form of medication or suggestion, are at last set down as hopeless neurasthenics or hysterics. The symptoms of which they complain are manifold, varying from attacks of simple indigestion to severe attacks of diarrhœa. The stomach symptoms are especially prominent in the young: attacks

[1] Read before the American Gastro-enterological Society, Boston, June 1, 1906.

of belching or pain at irregular intervals after eating, alternating with freedom from gastric distress of any kind, in spite of the most liberal diet. A very common symptom, also, is diarrhœa, especially in the morning, and freedom from the same during the rest of the day. Such attacks may occur daily, as I have seen in some cases; e. g., C. M., aged 37. Had no recollection of having had, at any time, appendicitis. Suffered for years with so-called "stomach trouble," which was characterized by diffuse cramping pains in the lower abdomen, followed by several evacuations of the bowels. Oftenest the pains were of such severity as to wake him out of his sleep, and only terminated after two or more bowel movements. These attacks came almost daily; the character of the stool was at first formed, then loose, and never occurred after breakfast. Finally, an attack coming in the middle of the day brought him to a surgeon, who diagnosed the case as appendicitis, and removed a swollen organ firmly bound down by adhesions. The patient, who had tried vainly medical treatment for years before, made a perfect recovery.

A similar case was Mrs. J. O., whose diarrhœas usually occurred daily from nine o'clock till noon; she ran the whole gamut of specialists, even to the point of having her round ligaments shortened, but finally recovered fully after an appendectomy.

In other cases the diarrhœic attacks occurred only at intervals, leaving periods of almost complete freedom from intestinal disturbances; e. g., A. F., a letter-carrier. Had five to six

movements, of slimy, mucoid character, accompanied by cramps, chiefly localized in the left hypochondriac region. Palpation disclosed a long swollen appendix, and deep pressure in the cæcal region elicited nausea and pain along the sigmoid. An operation was suggested after six months' medical treatment, combined with bowel massage. The appendix was swollen and firmly bound down by adhesions, but the rest of the bowel was free from all pathological changes. Patient now has only one to two formed bowel movements daily, in the face of a very liberal diet, whereas he formerly was hardly ever free from pain and diarrhœa, even when living on the most easily digestible food. In some cases (J. H. S. and Mrs. Dr. S.) the intestinal disturbance took on the character of a typical mucous colitis with small, hard scybala, often streaked with blood. In the first case the patient lost weight so rapidly that I deemed it advisable to have a long median incision made and the whole bowel explored for a possible neoplasm. Nothing, however, was found, except a long, almost obilterated appendix doubled over and adherent to the cæcum. Patient made a perfect recovery, and now has but one to two formed bowel movements daily, without cramps, and has begun to take on weight steadily. The second patient, who refused an operation, made a good recovery under dietetic and medical treatment combined with massage.

Often the cramping attacks are localized so low down in the abdomen as to be mistaken for diseases of the adnexa, and the unfortunate patients undergo tamponade and other local

treatment for months and years without results. In many cases, also, operations are performed to remove possible adhesions about the genitalia, but after the operation the patients continue to be the same sufferers they were before. Finally, after a long period of stomach lavage and bowel flushings, and dietings and starving, and electricity and massage, and sanitarium, the doctor puts his uncured and annoying patient in the convenient pigeonhole of neurasthenia, sets down all the symptoms as products of the patient's diseased imagination, and sits back complacently with that satisfactory explanation of his failure.

Ewald, in a paper presented before the Congress of German Surgeons in 1899, was the first to throw light upon this subject. He presented a number of cases which he termed collectively "appendicitis larvata," or masked appendicitis. The cases to which he referred present many of the obscure, unyielding symptoms which I have discussed above; none of the patients gave a history of appendicitis, yet a careful physical examination made it possible to diagnose chronic, subjectively unnoticed appendicitis, careful palpation disclosing a thickened swollen mass in the region of the cæcum. Ewald reports three cases of appendicitis larvata, which are of special interest.

CASE 1. Woman, aged 30 years; was previously treated for nervous complaints. She had pains all over the abdomen, occurring without assignable cause, and was compelled to stay in bed on account of weakness. Examination disclosed a cord of the size of a thumb, swollen, easy to palpate, and very sensitive to pressure, in the cæcal region. Recognized as processus vermiformis by means of inflation of the intes-

tine with air; cæcum swollen, succussion sound over the bowel, similar to that found with atony or paresis of the intestinal muscles. Anamnesis of appendicitis uncertain; operation. Appendix was found swollen, stiff, but not perforated; numerous adhesions to the vermiform process. Two flat tumors, with muddy, bloody, ulcerous contents, were found and evacuated. Recovery complete.

CASE 2. Male, aged 51 years; complained of constipation. For the last six months, continuous griping and drawing pains, especially in right region of the lower abdomen and below the umbilicus. The accompanying constipation was treated with aperients. Bowel evacuation showed some slime and blood. Inflation of the intestine disclosed a swollen appendix. At the operation the vermiform process was found attached to the fascia iliaca, and buried in adhesions The removed appendix shows no other irregularities. Recovery perfect.

CASE 3. Male, aged 30 years; anamnesis gave no indications of appendicitis. Patient had typhoid eight years before. Last summer, had frequent diarrhœa and considerable loss in weight. Examination disclosed no indication of disease of the stomach; chemistry of the stomach not especially disturbed. Local examination showed an elongated mass in the cæcal region and gurgling sound over cæcum. Operation revealed swelling and flexion of appendix, which contained hard fæces. Perfect recovery.

In conclusion, Ewald naturally says that appendicitis larvata, or perhaps I should substitute the term "ignota" as more appropriate, presents no characteristic symptoms, and that the diagnosis can be made only by excluding gall-bladder, stomach, and pelvic diseases, depending for the diagnosis upon the positive objective findings. At any rate, an operation in each case finally removed all the symptoms for good.

It is, however, not enough to promise a patient, who has submitted to an operation,

relief from his troublesome symptoms immediately after the diseased appendix has been removed. In my own personal experience, an operation seldom affords immediate relief. Often many months pass before the patient is finally completely cured.

Armin Huber of Zurich, in an elaborate article on appendicitis larvata, reports a number of cases, some of which are similar in character to those described by Ewald. It is interesting to note that others were mistaken for cholelithiasis, because there was pain and tenderness about the gall-bladder.

One or two of Huber's cases may be of interest.

CASE I. A man, aged 30 years; the appendicitis was mistaken for biliary colic. First attack came suddenly with violent convulsive pains in the region of the stomach, lasting two hours. The pains started at the pit of the stomach, and spread over the entire epigastrium, with no sensibility to pressure over the stomach. The day after this attack, patient felt well, and attended to business as usual. About ten months later, another attack of great severity. During the attack there were involuntary evacuations of the bowels; temperature 39° C. per rectum. One day he had convulsions that lasted several hours and then suddenly ceased. The temperature was normal by morning. Slight pains persisted for two days, after which subsidence of symptoms. Three weeks later a recurrence of the attack, with the old symptoms, and again sudden subsidence of the attack. All of the attacks were accompanied by severe vomiting. This time there were griping, drawing pains in the right hypochondrium, commencing at noon and lasting till 5 P. M. On walking, pains increased, and there was vomiting, rigor, and high temperature. In spite of morphine injections, pains continued all night; next morning the temperature was 39° C.; in the evening, 41.2° C. Fever lasted several days, then freedom from pain. About three months

later another violent convulsive attack occurred. The next day a tumor was observed *in loco classico*, distinctly discernible, supporting the diagnosis of appendicitis. The perityphlitic tumor disappeared in a few days, with rapid recovery; thirteen days later, operation. The appendix was directed downwards, 5 cm. long; slightly adherent to peritoneum, pelvis, and ileum. It was 3.5 cm. thick. Recovery complete. The first diagnosis in this case was cholelithiasis, because the anamnestic statements pointed to this, and because of the sensibility to pressure in the region of the gall-bladder.

CASE 2. A man, aged 32 years; the appendicitis again was mistaken for biliary colic. There were violent "stomach cramps." Yet on the next day the symptoms disappeared, and patient attended to business as usual. Probable diagnosis of biliary colic was made. About ten months later, another attack, but this time with certain sensibility to pressure over McBurney's point and the stomach. Second diagnosis, supported by a careful anamnesis: appendicitis simplex. Patient claimed that he had similar attack ten years before, though a slight one. Two years after, third attack — always with the same symptoms: pressure, pains in the stomach, no fever. A fifth attack lasted seven hours, and a sixth twenty-two hours. Interim. Operation later. The extirpated appendix showed strong catarrhal changes, with a shiny, bloody contents, and obliteration in the center. Complete recovery.

CASE 3. A man, aged 40 years, in whom cholelithiasis was mistaken for appendicitis; complained of violent pains in the region of the stomach; every time he took liquids the attack came on; no emesis or icterus. The examination of the abdomen showed sensibility to pressure in the right fossa iliaca. Diagnosis: appendicitis simplex. Operation recommended, but not performed, and a few weeks later patient was struck with typic hepatic colic, with high-grade icterus. This shows that in some cases a differential diagnosis between cholelithiasis and appendicitis is impossible.

CASE 5. A man, aged 40 years, who suffered for a long time with pain in the abdomen and stomach. The pains were of a griping character, localized mostly in the

epigastrium and in the right hip. The bowels were irregular and constipated. Examination revealed pronounced sensibility to pressure in the right half of the epigastrium. Patient left without treatment. It was learned later that he was stricken with perforative appendicitis. Operation twelve hours later. Death from general peritonitis.

CASE 6. A man, aged 39 years; complained of pains in the stomach, with dyspnœa; abdomen slightly distended and sensitive to pressure; spontaneous pains in the entire epigastrium. Region of the cæcum not painful to pressure. Attacks began with violent pains in the stomach; not severe enough to keep patient at home. However, the general condition became worse, and the patient was compelled to take to bed. A diagnosis of gastric catarrh was made. An exploratory puncture on both sides brought a light, bloody, thick fluid; perhaps a little pus. When transferred to the hospital, he had strong, offensive diarrhœa. The peritonitis symptoms increased. He died ten days after the beginning of his complaint. The autopsy disclosed appendicitis perforativa. In this case the symptoms of pleuritis were so marked that in the hospital the diagnosis was confirmed as pleuritis.

Similarly, Roth of Prague reports two cases of appendicitis larvata which do not present any special features which would warrant my reading them here, except that the diagnosis was only made post-mortem.

Often there are intractable cases of constipation, especially the spastic variety, which are due to an inflamed appendix. Ewald cites a case in which the patient's obstipation yielded to no medical or local treatment. She was treated for years for hysteria, even though, especially during her pregnancies, she had attacks very much like intestinal obstruction, characterized by cramping pains, bloating, and increased peristalsis. Inflation of the bowel showed localized pain and tenderness

over McBurney's point, and the operation a long inflamed appendix, adherent to the bowel and right ovary. In this case the operation brought permanent relief from the atony of the bowel and neurosis.

Similarly in one of my own cases, stubborn constipation and extreme neurosis yielded after an appendectomy. Often the pathological changes in the appendix in appendicitis larvata are so slight as to make it difficult to understand why there should be such pronounced subjective symptoms in these cases; yet if we compare them with the pain incident to unloading the bowel and bladder in women who have either adnexal disease or post-operative adhesions, the symptoms are much more easily appreciated.

Karewski of Berlin, two years previously, discussed cases similar to those reported by Ewald, in which the symptoms were variously interpreted as intestinal atony or obstruction. He failed utterly, however, to recognize the cause, and allowed his cases to come to perforative appendicitis. He reports two cases with stomach symptoms similar to those thus far read, and which I will therefore merely refer to at this time, except as an additional warning to keep appendicitis in mind in obscure stomach cases.

CASE I. A man, age not stated. The case began with obstruction and meteorism, eructation of gas, nausea; quick recovery. Two months later, renewed attack, with the same symptoms, but in a severe form; the patient could attend to business. A month later became bedridden. Bowels almost completely obstructed. Diagnosis: intestinal atony. A week later,

exitus, after operation for peritonitis. Operation disclosed a perforated appendix.

CASE 2. A child, aged 2 years. Two tumors below the umbilicus, on both sides of the bladder; extremely weak and poor; intense meteorism; temporary diarrhœas, followed by constipation. Operation revealed perforated point of appendix resting in a small abscess. Exitus twenty days after operation. Post-mortem showed the processus vermiformis as well as the cæcum covered with tubercular ulcerations, but no intestinal tuberculosis. The rest of the abdominal cavity was divided off from the tubercular nests by an adhesive peritonitis. The intestinal loops were agglutinated, but nowhere was found pus or ulcerative changes. The indications are that the case began as perityphlitis tuberculosa leading to peritoneal tuberculosis.

A point in diagnosis not clearly recorded in any of the cases I have gathered I have found several times as follows: For example, one patient complained of gastric symptoms, bloating, sense of fullness and weight after eating, with vomiting, especially during the menstrual period. Examination of the stomach contents showed slightly increased amount of free hydrochloric acid, otherwise chemically and mechanically negative. Bowels constipated. Loss of weight. Deep pressure over the stomach caused no distress; on the other hand, pressure over the ileocæcal region at once elicited the nausea, belching, and fullness of which the patient complained, without causing any tenderness to be felt over the appendix itself. Patient had an indistinct recollection of some attack in the lower bowel years before, but nothing definite. Deep palpation disclosed a thickened appendix. Its removal caused the subsidence of all the symptoms. Another patient (B. F.), who had chronic

diarrhœa for years, and in whom the appendix could readily be palpated, felt pain in the sigmoid region when pressure was made over the cæcum, combined with desire to evacuate the bowels.

In still another patient, E. M., aged 24, who always was in robust health, there were a number of attacks, characterized by pain in the epigastrium and vomiting, followed by a subsidence of the symptoms. Finally, during the World's Fair, there was a severe attack of intractable vomiting, accompanied by cramping pains in the lower abdomen, and on the second day diarrhœa. The attack was then attributed to the St. Louis water. Last Christmas day she was seized with a vomiting attack of such great severity that morphine had to be resorted to to check it. There was at that time no pain in the abdomen. The following day she was again seized with pain and renewed vomiting, and feeling of diffuse soreness over the whole abdomen. Temperature was 101.2°; pulse, 120. The tentative diagnosis of perforative appendicitis with general peritontitis was then made, the blood count running up to 30,000. The symptoms gradually subsided, and a week after the attack there was an easily palpable tumor in the ileocæcal region. Two or three days later there was a sudden evacuation of foul-smelling pus through the vagina, followed by great relief to the patient. The blood count went down to 8,000, and the patient was finally sent home, having refused an operation at that time. The operation, performed a month later, revealed two large abscesses in the abdomen. The appendix was bound closely down

to the cæcum and practically obliterated. Since then the patient has been in perfect health. In this case the attention of the attending physician, until the last attack, was never referred to the appendix. Her attacks were diagnosed as gastritis. This again shows the importance of considering the appendix as the seat of disease in all cases of periodical vomiting, especially when there are long intervals of freedom from any gastric distress in between.

Sometimes appendicitis larvata is taken for "growing pains," as in the case of M. K., who complained of pain in the right side, especially after playing hard or running. When palpation over the appendix disclosed the true state of affairs, the doubting parents could recall several attacks of spoiled stomach, one of which was accompanied by fever and vomiting. An operation in this case, as in that of Lucille P., a little girl, aged 6, who had violent stomach cramps while playing, substantiated the diagnosis and effected a cure. Similarly, Kelly and Hurdon report two similar cases.

CASE 1. Man, aged 27 years, with neurasthenia of long standing; colicky pains and temporary obstruction; frequent similar attacks every two or three months. Examination disclosed no symptoms of appendicitis. The pains concentrating during the attacks in the region of the pylorus and umbilicus; no sensibility to pressure in the region of the cæcum; some emesis. Diagnosis: hypersecretion, with hyperacidity and pylorospasm; finally, violent colicky pains in the abdomen led to the suspicion of appendicitis. Operation revealed a chronic ulceration of the appendix, with considerable induration and several tumors, of which one was near eruption.

CASE 2. Woman, aged 32 years; suffering for six years from cramping pains in the abdomen and ob-

stinate constipation, together with loss of flesh. The disease was diagnosed as genital. Then sudden, strong peritoneal symptoms, with collapse, came on, with fever. Operation revealed chronic appendicitis, with extended old adhesions in the region of the cæcum.

In view of the importance of successfully palpating the appendix in all cases of suspected appendicitis larvata, it will not be amiss to discuss the best methods of so doing. While I do not believe with Hausman and Edebahls that even the normal appendix can be felt in almost all cases that are favorable for palpation, yet it is undoubtedly true that in subjects that are not too stout, and who learn to relax sufficiently, the thickened and diseased appendix can very often be felt; and, what is very important, even if the abdominal walls are too thick to admit the palpating hand deeply enough, it is nevertheless frequently possible to elicit the symptoms which arouse the suspicion of chronic appendicitis by deep pressure. It is, of course, necessary to exclude a spastic bowel and other surrounding structures, but with great care and practice it is frequently possible to diagnose otherwise hidden appendicitis. In hysterical subjects, too, right-sided ovaralgia could readily be mistaken for inflammatory conditions about the cæcum, so this rather common condition must also be carefully eliminated.

If the patient does not relax sufficiently, lowering the head and partially flexing the right thigh often are of great assistance. In some cases examination in a warm bath is very useful. Ewald's recommendation to inflate the bowel seems to me more likely to cause con-

fusion than assistance in palpating the appendix. We must carefully keep in mind, too, that there are a number of conditions which could readily mislead the physician into making a false diagnosis of appendicitis, such as distention of the ileocæcal region with gas, circumscribed contraction of the rectus muscle, and colics due to intestinal parasities, besides the careful exclusion of adnexal and gall-bladder diseases.

It must also be borne in mind that there is often good effect from an appendectomy in which a normal appendix is removed. This, according to Kuttner, is probably due partially to suggestion, partially to rest and proper diet, and in some measure, perhaps, to the division or certain nerve trunks. There is, however, a much more potent reason than the ones just enumerated for the wonderful effects following the removal of a diseased appendix; for whereas these might hold in conditions simulating appendicitis, yet there are real anatomical reasons for the favorable effects cited in the cases quoted.

Lennander has shown that most of the pain in peritonitis is due to an accompanying lymphangitis; that the adhesions cause pain only when put on stretch, through distention of the bowel. This explains the cramping pains incident to chronic appendicitis, especially when the bowel above is filled with gas or stool. The lymph-vessels of the appendix do not press upon sensory nerves until the lymphangitis extending from the appendix reaches the perilymphatic tissue and lymph-glands surrounding the aorta. This also explains the

early localization of pain about the epigastrium in appendicitis. The pains in the region of the appendix only come later, when there is rubbing between the parietal and visceral peritoneum. Analogously, Kraft reports a case of left-sided appendicitis in which the attack began with pain in the epigastrium and vomiting, spreading some hours later, and localizing in the left lower abdomen and lumbar region. The post-mortem showed left-sided gangrenous appendicitis. It is therefore with good reason that Lenzmann warns against having the attention detracted from the appendix by general though not especially characteristic pains in the abdomen. There may be pain about the umbilicus, emesis and cramps, with a diseased appendix which causes no subjective symptoms to the patient even on deep palpation, yet an operation always shows characteristic changes. The projecting pains in appendicitis larvata are probably due to the anastomosis of the inferior mesenteric plexus with the plexus solaris. As Müller of Aachen suggested, after hearing Lenzmann's paper, the pain is sometimes due to a lymphangitis ascending to the ribs. It is a common occurrence to find that pleurisy begins with cramping pains in the abdomen and vomiting. So in appendicitis, we may find the reverse true. Yet in appendicitis, as in cholelithiasis, ureteral colic, etc., the subjective symptoms of the patient are a poor guide to the physician in the correct localization of the disease. An important diagnostic point emphasized by Nothnagel is stiffness of the rectus and increased peristalsis above, plus the objective findings.

In many of my cases there was found complete obliteration of the appendix; in others there were several strictures without complete destruction of the lumen of the organ. The symptoms in no way were commensurate with the morbid changes in the diseased organ, and often the greatest distress and discomfort occurred in cases in which the appendix showed very slight changes.

Dr. O. L. Schmidt kindly furnishes me with reports of three cases of appendicitis larvata, in one of which, although there were attacks of intense pain of a colicky nature, deep in the epigastrium and back, with belching, and in which the physical examination was absolutely negative, yet an exploratory laparotomy held five months after the last of the attacks showed such slight adhesions around the appendix, that it was hardly convincing to the operator that the real cause of the colics had been found, but, nevertheless, the patient made an uneventful recovery without any recurrence of pain for over a year.

In my own cases, also, I have found the most pain was present when there were the most adhesions, especially when the appendix was doubled over the cæcum and completely bound down. This class of appendix was oftenest found in cases of mucous colitis and diarrhœa with cramps; whereas stomach symptoms were oftenest associated with a long thickened appendix, which hung freer in the peritoneal cavity. This kind of appendix is naturally also the most dangerous, and can

readily perforate, whereas the string-like, narrowed variety is more annoying from its reflex symptoms than liable to lead to death from general peritonitis.

While no claim can be made that appendicitis larvata is a disease for itself, and with the full knowledge that it is merely a name for unrecognized chronic appendicitis, yet it is necessary to know that there are many appendicites taking atypical forms, the failure to recognize which is often responsible for the death of a patient who might otherwise have been saved by a correct diagnosis and operation. We are all too prone to call diseases we cannot understand "nervous" diseases, for want of a better term. A more honest name for some of them would be "diseases of which we do not know the cause." Appendicitis larvata and cholelithiasis account for many so-called nervous dyspepsias; but, perhaps, as our knowledge broadens we may be able to account for the rest. Malingerers and neurotic individuals may enjoy the distinction of having various ailments, but the fewest want to have their digestion interfered with and the food they like best forbidden them for the glory of being sick; besides, a person would have to be very neurotic indeed to find real enjoyment in cramps and diarrhœa. We must try to keep clearly in mind the important fact that we may do patients an injustice; that if we are not sure that the symptoms of which the patient complains are purely nervous ones, we may always satisfy ourselves by the relatively harmless procedure of an exploratory laparotomy, and thereby save the patient an intense amount of

suffering without endangering his life. The good internist is the one who best sees his limitations. It in no way detracts from the glory of the medical man if he adds to his diagnostic skill the mechanical aid of the surgeon.

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DISCUSSION.

Dr. A. L. Benedict, of Buffalo: It occurs to me that the condition which Dr. Kohn has described in his paper is possibly one to which the diagnosis of hyperchlorhydria is often given. In from fifteen to twenty per cent. of the cases of chronic gastric catarrh and chronic colitis there is a palpable appendix or a distinct area of tenderness in the appendicular region.

I believe myself that there is such a thing as typhlitis. We should always bear in mind the fact that the appendix is really an unexpanded portion of the caecum, and there are many cases of chronic inflammation of the bowel where the appendix is taken out and the symptoms still persist, just as we see certain pelvic symptoms persist after curettage of the uterus. If you have to deal with a typhlitis, you will not cure the condition by removal of the appendix.

The method of treatment which I have found very satisfactory in those cases is to make the patient practically a vegetarian, give salacetol or some similarly acting drug internally, and apply the Credé ointment externally, and in the course of three or four months I usually get a permanent cure.

Several years ago I tried the mineral oil treatment in these cases, but I found that the oils on the market contained too much kerosene. I then had a pure paraffin oil (known as Purpetrol) prepared especially for my use, which served as a lubricant and laxative, and also as a base for the administration of bismuth or some mild antiseptic, which to a certain extent at least checked the fermentative and putrefying changes in the intestines, and exerted a beneficial effect on the disease. In the treatment of these cases, the condition of the rectum should not be overlooked.

Dr. S. J. Meltzer, of New York City: There is no doubt that there are cases of so-called masked appendicitis which might be termed appendicitis larvata. I would like to say however, that if an abdominal pain disappears after an appendectomy it should not be taken for granted that an appendicitis had existed. All of you, no doubt, have seen cases of supposed intra-abdominal malignant growths which a laparotomy showed to be apparently inoperable and yet the patients improved after the operation and remained in excellent health for a number of years. Some twenty years ago, upon the strength of a false diagnosis, the first operation for a tubercular peritonitis was done. The patient recovered, and since that time laparotomy has been an accepted method of treatment in tubercular peritonitis, although no one knows why it benefits these patients. There are other conditions in which an exploratory operation is apparently beneficial, but we are still ignorant of the true significance of an exposure of the intra-abdominal organs. The profound effects of such a procedure are well shown in experimental work which we are carrying on now on rabbits. Any one

who has experimented with rabbits knows that the caecum of the rabbit, lying, as it does, on top of the intestines and largely covering up the rest of the abdominal contents shows no motion whatsoever. In reviewing the literature on the subject, I did not find it recorded anywhere that any one had ever seen peristaltic movements of the rabbit's caecum. We have since learned that under normal conditions, the peristaltic action of the caecum of the rabbit is even more pronounced than that in other sections of the intestinal tract, and that the absence of peristalsis of that organ, in the operated animals is due to the opening up of the abdominal cavity. The same applies to the active movements of the rabbit's stomach.

I only mention this as an indication of the profound influence which a mere abdominal incision may have upon the intra-abdominal organs. I believe that what has been observed in rabbits has its analogy, to a certain extent, in the human being, and that a simple abdominal incision may have considerable influence upon the future condition of the abdominal viscera, and that perhaps this happens also after removal of the normal appendix.

In the class of cases reported by Dr. Kohn, it is possible that not the appendix alone is responsible for all the reflex symptoms, but rather that it is only one link in a long chain. I dwell upon this point in my paper on Gastric Colic.

In connection with this general subject, I would again like to call attention to a method which I have found of great assistance in palpating the lower abdominal organs, namely, retraction of the psoas muscles. In some cases of appendicitis and sigmoiditis, another helpful sign is the referred pains which as a rule are confined to the side which contains the seat of the lesion.

Dr. John A. Lichty, of Pittsburg: I am much interested in Dr. Kohn's paper, because it really covers a very important question in reference to right-sided abdominal pain in cases where the lesion is of a doubtful character and where we hesitate to take radical measures for the relief of patients who are thus left in a more or less uncomfortable condition. I have in mind the case of a girl of sixteen or seventeen who suffers from occasional attacks of right-sided abdominal pain. These attacks sometimes come on at the time of menstruation, and sometimes in the intervals. She never had a frank attack of appendicitis. Her attacks are unaccompanied by fever or leucocytosis, but the patient feels uncomfortable. What should be done for such cases? Shall we advise operation? I have had a number of cases of this kind, and have found it difficult to come to a decision regarding them, especially if the patients are young, and living at boarding-schools or away from home.

With reference to Meltzer's sign for palpating the appendix, I have used it ever since I first heard him mention it, and have found it very helpful.

Dr. Kohn: If you take an appendix that has a number of strictures in it, and imagine the fæcal current trying to get through that appendix, you will have a good anatomical reason for cramps. If you add to that the possible presence of adhesions, the various symptoms that these patients complain of can be still more readily explained.

Extracted from the American Journal of the Medical Sciences, January, 1907.

GASTRIC ULCER IN CHILDHOOD.

By Harry Adler, A.B., M.D.,
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The following case of hematemesis occurring in a child of eight years has been of considerable interest, on account of its apparent rarity, and caused me to study the subject of gastric ulcer in childhood:

B. L., white, female, has been attending school, and living under excellent conditions of sanitation and nutrition. The family history is negative. The child was breast-fed, and well nourished in infancy. At the age of two years she had a rather severe attack of acute gastritis attended with fever and persistent vomiting for several days. She also had German measles, but otherwise none of the infectious diseases of childhood. For several years she has been "taking cold" easily, having several attacks of bronchitis every winter.

Her present trouble is of about ten weeks' duration. She suffers with repeated attacks of pain in the epigastric region. The pain occurs in paroxysms both before and after meals. Often it begins before meals, is relieved for a short time by taking food, and then returns sharper than before. It persists then a varying time, from one to three hours. At times it comes on in the midst of a meal. Sometimes it disappears quite suddenly. While in my office on the first examination she suddenly became nauseated and vomited, without apparent effort, food streaked with blood. The vomited matter gave the reaction for free hydrochloric acid. One week later, during an attack of pain, she again became nauseated and vomited about an ounce of bright blood.

The patient is rather frail and pallid. She weighs 42 pounds. The lungs and heart are normal. There is marked tenderness to pressure in the epigastrium. The stomach area is normal on percussion. The liver dulness is normal. The spleen is not palpable. There is no tenderness over the rest of the abdomen.

Examination of the blood revealed hemoglobin, 72 per cent. (Gowers); red cells, 4,200,000; whites, 8200. The urine was normal. The stools after the first appearance of blood in the vomit showed

blood with the turpentine-guaiac test. After the second appearance occult hemorrhage persisted for four days. Subsequent examinations failed to show its presence.

The patient was put to bed on a five weeks' ulcer cure. All food was withheld for thirty-six hours, after which diluted milk and bismuth were given. The stomach symptoms disappeared after the first three days, and remained absent during the entire course of treatment. Since then, a period of five months, the child has been free from symptoms and has gained steadily in weight.

From the symptoms, the attacks of gastralgia, epigastric tenderness, hematemesis, the presence of blood in the stools, and the result of treatment, the diagnosis of ulcer of the stomach is, I think, justifiable.

A study of the postmortem statistics shows that ulcer in children is absolutely and relatively of uncommon occurrence, as compared with its incidence in adults. Thus, in Welch's[1] collection of 607 cases of gastric ulcer, but one occurred in a child less than ten years of age. Lebert[2] found but one case in his series of 226. By Rehn[3] we are told that Rokitansky never encountered a case under fourteen years, and was himself, in 1873, able to collect but 9 cases in children. Fenwick[4] collected 18 cases, and Cutler[5] in 1904 added 11 cases. It must be remembered that these statistics have been gained largely from hospitals where postmortem examinations on children are comparatively infrequent. An examination of the statistics of children's hospitals shows a somewhat different result. Dusser[6] found ulcer in the stomach in 9 cases; and ulcer of the duodenum in 4. In 390 autopsies at the Babys' Hospital in New York, Martha Wollenstein[7] found 5 cases of gastric ulcer. Holt[8] reports 3 cases accompanying follicular gastritis and 5 accompanying general tuberculosis. In order to obtain data as to its occurrence clinically, besides consulting the text-books on diseases of children, I wrote to the leading pediatrists of this country, with the following results: Booker, Rotch, Griffith, Louis Starr, and Koplik have never seen a case that they recognized clinically. Koplik[9] reports one case seen at autopsy accompanying empyema, which he states represents his experience covering fully 300,000 cases of diseases in children. Jacobi,[10] on the other hand, states that it is not quite uncommon in children from seven to thirteen years. From the statistics of the

[1] Pepper's System of Medicine.
[2] Krankheiten d. Magens, 1878.
[3] Jahrb. f. Kinderheilkunde, 1873, p. 19.
[4] Disorders of Digestion in Infancy and Childhood, London, 1897.
[5] Transactions Amer. Gastroenterolog. Soc., 1904.
[6] Thèse de Paris, 1890. Cited by Holt, Diseases of Children.
[7] Cited from Osler, Practice of Medicine.
[8] Diseases of Children and Personal Communication.
[9] Ibid.
[10] Diseases of Children.

children's hospital it appears that it occurs oftener than it is recognized clinically.

VARIETIES OF ULCERATION. Fenwick recognizes five varities of gastric ulcer: (1) Hemorrhagic erosions, (2) follicular ulceration, (3) tuberculous ulcers, (4) malignant ulcers, and (5) simple ulcer. The last may be either acute or chronic.

(1) *Hemorrhagic extravasations* into the gastric mucosa are frequently noted in children, as in adults. They are observed as the result of congestion of the vessels of the mucosa, secondary to uncompensated valvular disease or to dilated heart, complicating chronic pulmonary trouble, or more rarely in cirrhosis of the liver. They have also been seen in the hemorrhagic diseases, as purpura, scurvy, leukemia, hemophilia, and in jaundice. The hemorrhages vary from minute points, barely detectable by the naked eye, to irregular oval or linear areas. The smaller are limited to the mucosa, while the larger effusions may involve all the coats. This condition is of pathological rather than clinical interest. As will be seen later, Kundrat[11] considers the true ulcers to have their origin in the necrosis of the hemorrhagic areas.

(2) *Follicular ulceration* occurs for the most part in infants as the result of follicular gastritis. According to Fenwick: "Here and there small hemispherical masses about the size of a pin's head may be observed upon the surface of the mucous membrane. These appearances are due to inflammation of the solitary lymph glands situated in the wall of the stomach, and it is from the necrosis of these structures that the ulcers are produced. The swollen gland presses upon the gastric tubules which surround and cover it in, and gradually causes destruction of those in the immediate vicinity. A minute abscess is formed which opens on the surface." The disease seldom extends deeper than the submucous coat, but occasionally the muscular and the serous coats become involved.

According to Holt, these ulcers give rise to no symptoms other than those of the attendant gastritis, as a rule; he has, however, seen three fatal cases from hemorrhage occurring in young infants. The ulcers in these cases resembled those found in the intestines in follicular enteritis.

(3) *Tuberculous Ulcers.* Ulceration of the stomach due to tubercle is apparently less rare in children than in adults. Steiner and Neureutter met with 4 cases in 302 autopsies on tuberculous children. Widerhofer noted it twice in 418 cases, while Holt reports 5 cases in 119 autopsies in tuberculous children, but states that there is no positive evidence that the ulcers were the result of tubercle. Together this is a total of 11 cases of ulcer in 839 cases, as against Durk's[12] findings of but 4 cases in 900 autopsies on tuberculous subjects.

[11] Gerhardt's Handbuch der Kinderkrank., 1880.
[12] Cited from Hemmeter's Diseases of the Stomach.

(4) *Malignant ulcer,* or rather the occurrence of carcinoma on the basis of a gastric ulcer in childhood, is unknown.

(5) *True peptic ulcer* in children is either acute or chronic. Morphologically they do not differ essentially from those seen in adults. Kundrat states that recent ulcers differ from those in adults only in that they are not always round, varying from a hempseed to a lentil in size. Their pathogenesis is as little definitely understood as in adults, though certain of the causes which are given to explain their occurrence can hardly be accepted as operable in children; such as hyperacidity and degeneration of the bloodvessels of the mucosa. Kundrat considered them the result of hemorrhagic effusions in the mucosa, destroying its vitality and permitting autodigestion by the gastric juice. He says: "If, as is commonly considered, hemorrhagic infiltration is the cause of hemorrhagic erosions, then for ulcus rotundum the same cause must be accepted. The difference between them is quantitative only and not qualitative. In the former case only the mucosa is affected, while in the latter the gastric juice acts also upon the submucosa and muscular layers." In support of this view may be cited the fact that hemorrhagic erosions and true ulcers are often found together in the same stomach. Ewald[13] inclines to this for at least a portion of the cases. Gerlardt[14] furnishes examples of erosions changed into true ulcers.

It is difficult to explain the occurrence of ulcers in the first few days of life on any basis other than that during parturition a hemorrhage into the mucosa has taken place and been followed by autodigestion. An infectious origin in children is indicated by the occurrence of gastric ulcer in children dying of tuberculosis, typhoid fever, pneumonia, noma, diphtheria, and empyema (2 cases) Ewald looks upon the bacteria as causal in a mechanical rather than toxic manner. He cites the observations of Nauwerk, Letulle, Dieulafoy, Stokes, and Kraft, who found emboli consisting of masses of bacteria closing up the smallest vessels of the mucosa. These are supposed to give rise to circumscribed disturbances in the nutrition of the mucosa followed by necrosis.

One acute case was evidently of traumatic or infectious origin following severe burns of the arms and legs.

Site of the Ulceration. With the addition to Cutler's statistics of several recent cases in which histories could be obtained, acute ulcers are found as often at the cardia as at the pylorus. They were midway between the two openings in two, and generally distributed in two. In chronic cases there appears also to be no special predilection of site. Multiple ulcers widely separated are frequent.

Symptoms. These have been obtained from only some of the cases, the reports being inaccessible in some cases and deficient in

[13] Deutsche Klinik, p. 459.
[14] Virchow's Archiv., Band cxxvii, p. 85.

others. In infants gastric symptoms are as a rule absent, indefinite, or masked by those of an infectious disease, a sudden hemorrhage, or pain from perforation, being the first evidence of the presence of ulcer. In a number of cases the ulceration was an unexpected pathological finding. In older children the symptoms are similar to those in adults, but seem to be milder. Excepting immediately preceding perforation, in but one case was the pain severe.

Pain and tenderness were noted in but 13 per cent. of the acute cases; but were present in over 60 per cent. of the chronic cases. The pain was usually not severe. In those cases in which the patients were able to impart information, it was localized in the epigastrium. In young children it seemed over the entire abdomen. In all cases it came on in paroxysms, occasionally before, but more usually after meals, and was relieved by vomiting. Intense pain was followed by hematemesis or perforation.

Vomiting. In the acute cases in infants. vomiting was usually subsequent to diarrhœa. In older children it followed epigastric pain and relieved the distress. In cases accompanying follicular gastritis, vomiting was persistent. Fenwick mentions the finding of minute pieces of mucous membrane in the vomit in cases of gastric erosions.

Hematemesis was noted in 40 per cent. of the cases in which histories were obtained. It was bright red and varied from a few streaks in the vomited material to a quart(?). In 4 acute cases it was the immediate cause of death; the stomachs were found filled with blood. In chronic cases the hematemesis was preceded by epigastric pain. In the acute, pain was often absent.

Symptoms of Indigestion. Anorexia, fulness, and distension, nausea, or heartburn were seldom complained of. Excepting acute cases, they were noted only in the chronic cases.

Stools. Diarrhœa was a prominent symptom, being present in almost one-half the acute cases. It usually preceded vomiting. Constipation was at times followed by diarrhœa. Melena was noted in the majority of cases of hematemesis. I have not been able to find a case in which it completely replaced hematemesis, although it is mentioned as a possibility, by Fenwick. Occult bleeding was, with the exception of my case, not noted.

Fever was noted in a considerable number of cases, running in infants as high as 106°. Cutler reports a temperature of 102° in a girl of six years and 101° in a child of eight.

Blood examinations were made in but 2 cases. Both were anemic. Hemoglobin, 75 per cent., and 60 to 85 per cent. In one the leukocyte count was normal; in the other, 25,000 to 27,000.

COMPLICATIONS. A striking feature of acute ulcer in children is the large percentage of *perforations.* In 18 cases in which autopsies were made perforation was found in 6, or 33 per cent. In 8 fatal

cases of chronic ulcer it was present in 3, making a total of 9 cases of perforation in 26 fatal cases. The symptoms are sharp, sudden pain in the abdomen, vomiting and collapse. Death occurs within thirty-six hours. This complication is not infrequently the first indication of ulcer. In older children, symptoms of the complicating peritonitis are sometimes seen, as in adults; but in young children collapse with rapidly fatal termination is the rule. Bisset has recently reported a case of perforation in which the initial symptom was profuse hemorrhage. In 1 case, perforation of the diaphragm occurred with subsequent pyopneumothorax. In Chvotek's[15] case stenosis of the pylorus and dilatation of the stomach is recorded. The base of an ulcer had formed adhesions with the pancreas, and contracted narrowing the pylorus so that it would hardly admit the tip of the little finger. Krasnobayeff,[16] reports 3 cases of stricture of the pylorus in children.

Robinson[17] reports a case of chronic ulcer in a boy of fourteen years, in whom autopsy showed two strictures of the œsophagus with a diverticulum. The stomach was markedly contracted in size and the pylorus thickened and constricted. At the pylorus was a large ulcer almost encircling it. The mucosa was replaced by fibrous tissue to which the contracture of the stomach was probably due.

Diagnosis. In those cases in early infancy in which the first symptoms are profuse hemorrhage or collapse from perforation, early recognition is obviously impossible. In older children the symptoms are usually so little distinctive, and the disease of such uncommon occurrence that the diagnosis must depend largely on the finding of blood in the vomit and stool. And here care must be taken to eliminate the many other causes of hematemesis and melena. Thus in Dusser's 24 cases of hemorrhage from the stomach and intestines, in only 13 was ulcer found. In infants the blood may rise from the gums, nose, throat, or lungs, and be swallowed, followed by vomiting. Blood may be sucked from the breasts of the mother, or, in neonats, have been swallowed during parturition. True hematemesis arises from a number of causes other than ulcer. It has been observed in purpura, hemophilia, scurvy, and similar diseases in which there is a tendency to bleeding from any of the tracts. Cirrhosis of the liver, a not very uncommon condition in children, may give rise to it. In malignant forms of the exanthemata, notably measles and variola, it is likely to occur. I have seen it in a case of influenza. Other causes are: corrosive poisoning, severe retching (as may occur in pertussis), and traumatism as due to swallowing of foreign bodies. The great majority of these can be eliminated with a little care. In cases of melena the elimination of other causes

[15] Archiv. f. Kinderheilkunde, 1882, vol. iii, p. 267.
[16] Dietsh. Moskau, 1898, iii, p. 195.
[17] International Clinics, 1905, 15, s. iii, p. 119.

presents greater difficulties. It is present in practically all cases of hematemesis, either as visible or occult hemorrhage. The importance of its recognition lies in the possibility of it completely replacing the vomiting of blood. In addition to the causes already mentioned as producing hematemesis, the possibility of intestinal origin must be considered. These are ulceration of the bowel due to catarrhal enteritis or colitis, tuberculosis, and typhoid fever; further, intussusception, thrombosis of the mesenteric artery, intestinal parasites, rectal polyps, hemorrhoids, and straining at stool. In neonats, melena frequently occurs without discoverable cause.

It is not unlikely that, with the attention now being devoted to the recognition of occult hemorrhage, curable ulcer of the stomach will be more frequently recognized in children suffering with chronic stomach disorders.

DISCUSSION.

Dr. J. C. Hemmeter, of Baltimore: Dr. Adler's paper is a very valuable contribution to this important subject, and includes a good resumé of the literature. In this connection I would also refer to a recent contribution to the pathogenesis of gastric ulcer by a member of our Association who is not present, Dr. Fenton B. Turck, of Chicago. He attributes the occurrence of peptic ulcer to the colon bacillus.

Dr. Max Einhorn, of New York City: About three or four months ago I saw in consultation a girl of twelve years who was undoubtdely suffering from a gastric ulcer. There was a history of gastric disturbance of several month's duration, the symptoms gradually becoming more severe, and accompanied by a succession of gastric hæmorrhages. She was given the rest treatment and made a good recovery.

While the occurrence of gastric ulcer is rarer in children than in adults, the lesion is at times met with in early life.

RECENT STUDIES IN THE DIAGNOSIS OF GASTRIC ULCER.

By Dr. J. C. Hemmeter, of Baltimore, Md.

DISCUSSION.

Dr. A. L. Benedict, of Buffalo: A few years ago the assertion was made that the differential diagnosis between gastric ulcer and gall stones could be made by detecting the stones with the X-ray. I would like to inquire whether any one present has had any experience with that method? My own has been extremely unsatisfactory.

Dr. Hemmeter: The assertion that gall stones were visible by the X-ray was attributed to Senn. We gave the method a trial, not only in subjects who had gall stones, as shown by operation, but also with gall stones outside the body, and the results were entirely negative.

Reprinted from the New York Medical Journal for August 11, 1906.

THE SUBSEQUENT HISTORY OF THIRTY-TWO CASES OF CHRONIC GASTRIC ULCER PREVIOUSLY REPORTED.*

By Frank Hall Murdoch, M. D.,
Pittsburgh, Pa.

At a meeting of this association, held three years ago, it was my privilege to read a paper on The Treatment of Chronic Round Ulcer of the Stomach, based on thirty-two cases seen in private practice prior to December 31, 1902; and as a good deal has been written since that time regarding the incurability of chronic gastric ulcer by medical means, I thought it might be of some interest to you to know the subsequent history of these cases. Two of the patients referred to have since died; one from aneurysm of the aorta and one from hæmorrhage, the result of a recurrence of the ulcer. In February of this year I communicated directly with the survivors, either by personal interview, by letter, or through the family physician, and found that twenty were permanently cured, four greatly improved, three were not improved, and three could not be traced. All of the twenty patients who had been permanently cured were treated more than three years prior to February, 1906 and some of the worst cases four, five, six, and one seven years prior to that time. Of the four cases that were greatly improved, there had been, so far as I could ascertain, no return of the ulcer: but the patients did not consider themselves in good health. One of these patients, a year and a half after begin-

* Read at a meeting of the American Gastroenterological Association at Boston June 4, 1906.

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ning to eat solid food, suffered from a mild degree of ischochymia, which was relieved by medical means. I saw her in February of this year, and she was able, at that time, to take three meals of solid food each day, and maintained her strength and weight.

The proportion of these cases would be as follows: 66.6 per cent. permanently cured, 13.4 per cent. greatly improved, ten per cent. not benefited, ten per cent. not heard from. Fatal hæmorrhage occurred in 3.1 per cent., and in 3.3 per cent. there was a mild degree of stenosis of the pyloris.

And just here I would like to refer to a misapprehension under which some American surgeons are laboring in respect to Leube's views regarding the proper time for surgical interference in chronic gastric ulcer. For example, one writer quotes him as follows: "Even Leube, the most optimistic of medical men, says that gastric ulcers which are not cured in four or five weeks by medical means will not be cured by a longer treatment" (1). And another remarks that "Leube says that from four to five weeks should cure gastric ulcers which can be cured medically" (2). Thinking that in some way these surgeons had misapprehended Leube's views on this subject, I wrote to him in February of this year, and he honored me by replying as follows: "There must be a misunderstanding in the quotations referred to. I enclose my last treatise on *Ulcer of the Stomach,* of 1897, in which you will find on page 6 (marked with pencil) my opinion of the curableness of ulcer of the stomach by the well known cataplasm diet cure in persistent cases." The paragraph referred to, translated by a competent authority, is as follows: "From my point of view and on the basis of the preceding statistics, I wish to emphasize

that in by far the greatest majority, that is, in from seventy-five to ninety-six per cent. of the cases, surgical treatment is not even considered; in seventy-five per cent. of the cases surely not before a repetition of the treatment has proved the incurability of the ulcer by medical means. Usually those cases which have after the first treatment been only improved, or have even remained uncured, will completely heal up after a second or third application of the treatment. The principal thing for physician and patient is not to flag in patience and consistency. I have seen that such repetitions of the treatment, even in ulcers of ten years, and longer standing, worked at last a complete cure" (3)

Here then are Leube's views on this point at first hand, from which it appears that instead of recommending operation at the end of four or five weeks in case an ulcer of the stomach is not cured by medical means, he insists on a repetition of the same treatment, and shows that in from seventy-five to ninety-six per cent. of his cases this method of treatment results in a permanent cure.

References.

1. *Journal of the American Medical Association,* December 1, 1900, p. 1388.
2. *Medical News,* April 16, 1904, p. 724.
3. *Sonderabdruck aus dem Archiv für klinische Chirurgie,* lv, part 1.

816 Empire Building.

DISCUSSION.

Dr. G. W. McCaskey, of Fort Wayne, Ind.: The subject of the medical and surgical treatment of gastric ulcer seems to be in the air. At the meeting of our own State Society in Indiana last week we had a symposium on this subject in which particular stress was laid upon the proper limitations of the surgical treatment of gastric ulcer. In several of the sections of the American Medical Association the subject is also on the program for discussion.

I was glad to hear Dr. Murdoch's paper, and especially to hear at first hand the opinion of Leube on the feasibility and effectiveness of medical treatment in gastric ulcer which clearly shows that he has been misquoted. In the paper which I read on this subject last week I made the assertion that in about 80% of all cases of gastric ulcer, surgical treatment was not called for from any point of view. In the remaining 20% there was a certain proportion in which surgical intervention should be advised.

An important question in connection with this subject is the length of time during which the medical treatment should be carried on before surgical aid is invoked. Mumford & Stone, in their recent book on the surgical treatment of diseases of the stomach, condemn the statement of a certain surgeon that all cases of chronic indigestion of two years' standing or over were proper cases for surgical treatment. They are entirely correct as no such sweeping rule can be laid down. It depends on the sort of treatment that the case has had, although, that is perhaps begging the question. Each of us, no doubt, has a method of treatment which we think is just a little better than that of anybody else; but certainly, in any case of gastric ulcer, the treatment should be in the hands of a competent internist, and the patient should have the benefit of such treatment for a considerable time before surgical intervention is even considered.

I do not believe—contrary to what appears to be the prevailing opinion among surgeons today—that a case of gastric ulcer is any more completely or permanently cured by gastroenterostomy than by medical treatment. That point remains to be proven by the surgeons. I think there are other objections to a surgical operation in these cases. Adhesions between the stomach and adjacent viscera are apt to follow such operations, and the effect upon the intestinal functions is injurious. There are cases of gastric ulcer, of course, where a gastroenterostomy is imperative, and we must choose the lesser of two evils, but if the patient can be cured by medical means, he is just

as well off as after the surgical treatment so far as the permanency of the cure is concerned.

Dr. Murdoch: In the treatment of gastric ulcer by surgical methods, it is entirely useless to do a gastroenterostomy, unless a stenosis of the pylorus exists, as the experiments of Dr. Cannon have shown that, notwithstanding the presence of the artificial opening, the food will still continue to pass through the pylorus.

DEMONSTRATIONS OF GASTRIC AND INTESTINAL MOVEMENTS.

By Walter B. Cannon, in the Physiological Laboratory of the Harvard Medical School.

For the purposes of this demonstration, a cat has been anaesthetized, and after the destruction of its spinal cord up to the neighborhood of the phrenic nuclei, the animal was immersed in warm normal saline solution, and the abdomen opened. Gastric peristalsis, segmenting movements in the small intestine, and antiperistalsis of the proximal part of the large intestine can thus be directly observed and studied. The method of demonstration is that of Bayliss and Starling.

Antiperistalsis, which has been generally denied in the small intestine, is the normal movement in the proximal part of the colon. This activity of the large intestine, which was first observed incidentally by Jacobi in the opened abdomen, was established as the natural activity in the undisturbed animal by observations in 1902 in the Harvard Physiological Laboratory. The report of the experiments was published in the American Journal of Physiology, 1902, vi, p. 265. In 1904, Elliott and Barclay-Smith confirmed these observations, and also found in animals with a sacculated proximal colon that there were kneading and churning effects due to oscillating movements of the walls of the sacculi. All these observations on lower animals show that the most active part of the large intestine is the proximal part. The evidence from auscultation indicates that in man also the ascending colon is the region of most energetic movements. These churning movements, and the antiperistalsis driving the food into the closed caecum must again thoroughly mix the food with digestive secretions brought from the small intestine, and expose digested food to the absorbing wall.

DISCUSSION.

DR. S. J. MELTZER, OF NEW YORK CITY: Although the antiperistaltic movements of the large intestine were first observed by Jacobi, in 1890, his discovery was purely accidental, its significance being first pointed out by Cannon, in 1902. You can establish also in human beings by palpation, by a feeling of vibration transmitted to the fingers, that there is an antiperistaltic movement in the right half of the transverse and in the ascending colon.

DR. CANNON: The antiperistaltic movements continue as long as there is food going into the colon. They pass over the colon whenever a new mass of food enters the large from the small intestine. In animals in which the spinal cord had not been destroyed I have seen the movements going on fairly well, but not continuing for any length of time. They also cease when the animal is under the deep influence of ether.

In connection with the production of rythmic sounds by the stomach and intestines, my first observations concerning which were reported at the meeting of this Association in 1905, I would like to say that further investigations have confirmed my view that there is much greater activity on the right side of the abdomen than on the left. This is manifested by the more frequent occurrence of sounds in the former region. I was not able to hear rythmic sounds in the caecal region. As already stated, Elliott and Barclay-Smith, who have studied the activities of the colon, found that in animals with a sacculated colon, a churning activity of the walls of the sacculi was more prominent than antiperistalsis. As the human colon is sacculate it is probable that the churning activity occurs more frequently than antiperistalsis, and therefore rythmic sounds would not be produced.

RUPTURE OF THE COLON FROM VIOLENT PERISTALSIS PROBABLY INDUCED BY FECAL IMPACTION.

BY

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of Ft. Wayne, Ind.

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[Reprinted from *American Medicine*, New Series, Vol. II, No. 2, pages 91–93, February, 1907.]

RUPTURE OF THE COLON FROM VIOLENT PERISTALSIS PROBABLY INDUCED BY FECAL IMPACTION.[1]

BY

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Perforation of the intestinal wall, at some point weakened by a local lesion usually of ulcerative character, is a familiar fact in pathology. Such perforations, perhaps, usually occur at the moment when more or less strong peristaltic movements increase the intraintestinal pressure. In a rather careful search of the literature, I have been unable to find any reference to a more or less extensive tear or rent of the intestinal wall thus produced in the absence of a palpable lesion easily determined macroscopically.

The following case has, therefore, appeared to me to be sufficiently rare, if not unique, to justify a brief note concerning it, even though a proper study of the specimen was not permitted.

The patient, Mrs. H., aged 42, married, was referred by a physician from an adjoining town, March 18, 1905.

There was a history of chronic constipation with considerable gaseous eructations with headaches and other evidences of disorder

[1] Read before the American Gastroenterological Association, Boston, June, 1906.

of the digestive organs. Nine months prior to my examination, abdominal pain appeared as a troublesome symptom, starting in the left lower quadrant and traveling over the line of the colon in the reverse direction. These symptoms increased in severity and the peristaltic waves could be distinctly seen through the abdominal wall. The symptoms became so severe that a laparotomy was done by a surgeon three weeks before I saw the patient, for the purpose of relieving a possible partial obstruction due, perhaps, to inflammatory bands or adhesions, but nothing of this character was found. A salpingitis and diseased ovary were found and the tube and ovary removed but no relief was obtained.

The patient was sent home for a few weeks to allow the abdominal wound to become strong enough to resist the straining incidental to stomach examinations, when she returned for further investigations. Physical examination was substantially negative with the exception of slight enlargement of the liver. A test-meal showed a complete absence of free hydrochloric acid. There was no Gunsberg reaction although there was a little color with the dimethyl which was probably due to lactic acid, a small quantity of which could be determined by Uffelman's test. The fasting stomach in the morning contained considerable debris made up principally of mucus, glandular and epithelial cells.

The colon contents consisted largely of small round hardened masses of feces, considerable fine mucus, numerous leukocytes, starch cells and meat fibers being found microscopically. Analysis of the urine was negative with the exception of indican in moderate amounts. Hemoglobin was 70 percent with 9,000 leukocytes.

Excluding stenosis on the basis of a laparotomy, I expressed the opinion that the violent peristalsis of the colon was excited by the mass of fecal matter in its lower segments

which it was unable to propel onward. This would amount, in fact, to a temporary obstruction which was finally overcome by laxatives and enemas and the patient relieved until reaccumulation took place. This was indeed borne out to a large extent by the history of the case, the different exacerbations being associated with accumulations of fecal matter. The treatment instituted included the free use of strychnin hypodermically, the use of laxatives so far as required, supplemented by colon lavage, a carefully regulated diet and the local use of electricity.

There was considerable improvement in the course of one or two months and there seemed a fair prospect of entirely overcoming the trouble in the course of time. One evening, however, she ate an unusually full meal which was followed by symptoms of indigestion.

On the following morning the spasmodic pains already described and which were undoubtedly due to a violent peristalsis of the colon, became very severe. While walking in the yard she was seized with intense pain and passed rapidly into collapse and died in the course of a few hours before anything in the way of an exploration could be done.

The *autopsy* was made by the attending physician and I was unable to be present. At the splenic flexure of the colon, there was found a tear extending two or three inches in a longitudinal direction, through which the colon contents escaped into the peritoneal cavity with rapidly fatal result. A careful dissection of the colon below the laceration showed a distinct narrowing of the lumen of the tube amounting to a moderate stenósis, although it was not sufficiently marked to permit its discovery by manipulation at the time of the surgical operation. Within this narrowed portion and above it, there was considerable fecal matter producing a partial

obstruction at this point and the laceration was clearly the result of an extremely violent peristaltic wave in attempting to force the colon contents through this portion of the tube.

The attending physician was forbidden to remove any part of the intestine for examination but there was nothing in the nature of an induration or an ulcerative process or anything else which could be determined by inspection or palpation carefully made. There may, of course, have been some degenerative process which involved the muscularis and so far weakened the intestinal wall as to allow it to give way under the pressure. In the absence of a histologic examination such a possibility cannot be denied but so far as a macroscopic examination, made by a very careful, intelligent and conscientious physician could determine the colon wall at the seat of the laceration was normal in character.

Another case somewhat similar to this so far as the peristaltic movements is concerned was under observation for a short time.

The patient was a young lady of about 25, and the peristaltic movements were not especially painful but could be seen distinctly along the line of the colon in both the direct and reverse directions, associated with a violent churning of the contents giving rise to sounds which could be heard at times 75 to 100 feet away. This patient was extremely neurotic but also had a tuberculous history, one of her sisters having since died of tuberculosis. I advised the patient to remain in the hospital long enough to make a thorough study of the case with the view of possibly advising an exploratory laparotomy but she did not see fit to do so and disappeared from view.

Both of these patients were of a neurotic type and it is very probable that in both there was slight obstruction to the onward move-

ment of the colon contents and that the resulting violent peristalsis was in a large measure the result of indeterminable morbid nervous impulses.

In the fatal case which is the special subject of this paper, it would seem as though the laceration must have occurred in connection with the direct and not with the reverse peristaltic contraction. The failure to find the slightly narrowed portion of the colon during the operation emphasizes the difficulties of intestinal exploration by surgical procedures. In the absence of induration or marked distention of the proximal segments of the intestine the detection of moderate grades of stenosis must be extremely difficult and under certain conditions impossible. Such experiences show that the most scrupulous care should be exercised in examining the entire length of the colon and that the question should be seriously considered in each case as to the propriety of resection of a segment of the colon even if moderately narrowed, although, as in this case, fecal matter undoubtedly may pass the opening and under favorable conditions without difficulty.

DISCUSSION.

DR. A. L. BENEDICT, OF BUFFALO: While I have never seen a case similar to the one reported by Dr. McCaskey, I would like to refer to a rather unusual cause of rupture of the gut that has come under my observation. In certain foundries where radiators are manufactured, they use air blasts at high pressure, and last fall I saw an autopsy performed on a Pole who had been subjected to this high pressure through his clothing. The air-blast had entered the rectum, and had ruptured the bowel at the lower part of the splenic flexure with fatal results, Two similar cases have been reported in the lay press, one from Chicago, the other from a small town in New York. To all three, the lesion was fatal and, in all, the air was turned against the anus as a practical joke, without criminal intent.

In this connection it occurs to me to emphasize the possibility of intestinal rupture, from the introduction of water or air into the bowel through a tube the nozzle of which has not yet passed the anus. A stream of water at comparatively low pressure may overcome the force of the sphincter and enter the bowel.

DR. JOHN A. LICHTY, OF PITTSBURG: It is unfortunate that an autopsy was not permitted in Dr. McCaskey's case. I had a case two years ago in which there were tubercular ulcerations of the bowel, the colon particularly, with partial constriction at the splenic flexure. I am inclined to believe that a careful post-mortem examination in Dr. McCaskey's case would have revealed an ulcerative lesion in the region of the strictured gut.

In my case, the patient gave very definite symptoms of partial obstruction, and I was able to feel the induration in the bowel. The condition was confirmed at autopsy.

DR. S. J. MELTZER: Perhaps some who have read my article on colic will remember my definition of it, which is as follows: If there is a strong contraction for one reason or another at a certain point of the intestine, while a strong peristaltic wave progresses toward that point, the increasing distention of the section of the gut between the contracted points is the cause of the pain and it might, in some cases, be so great as to even produce rupture of the gut. Cases of the type reported by Dr. McCaskey had been described in literature, and in seeking an explanation for them, it is not necessary to invoke the aid of an organic lesion. A small section of the gut into which a certain amount of air or faecal matter is compressed by two opposing forces must rupture if the pressure is not relieved, and that can occur without the presence of an anatomical lesion.

DR. C. D. SPIVAK, OF DENVER, COL.: It seems to me that rupture is more liable to occur in the absence of an organic lesion, because when such a lesion is present, the consequent narrowing, if it occurs at all, will come on gradually, giving the intestine time to accustom itself to the abormal conditions, whereas, if an undue amount

of pressure comes on suddenly as the result of an acute condition, a rupture of the gut is more liable to occur.

Dr. McCaskey: I think an ulceration of the bowel can be fairly excluded in this case, and that its assumption is quite unnecessary. The patient was not undergoing any violent exertion at the moment of this rupture; she was standing or walking quietly, when she was suddenly seized with a violent pain; she was assisted into the house and went into collapse.

As to the nature of this narrowing of the gut I cannot of course speak positively, but from the description that was given me it was just such a narrowing as I have seen several times at autopsies, and without having been at all important. The surgeon who did the laparotomy said he could neither feel anything nor see anything, so, as I stated in my paper, I think that both a neoplasm and an ulcerative process can be fairly excluded. The only thing that I think cannot be excluded is a degenerative process of some sort; to discover this it would have requird a histological examination, which was not permitted. Perhaps the narrowing of the bowel in this case was a congenital condition.

Dr. Meltzer's explanation of colic is very interesting. Of course, the colicky pains were produced by the direct and not the reverse peristaltic waves.

[Reprinted from *American Medicine*, New Series, Vol. I, No. 5, pages 249–255, August, 1906.]

ON THE INFLUENCE OF REST, EXERCISE, AND SLEEP ON GASTRIC DIGESTION.[1]

BY

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The question concerning the influence of rest and exercise upon the digestion of food has at all times attracted much attention; even the older writers have commented upon this subject. According to Hippocrates,[1] "Rest greatly aids digestion" and there is an old latin saying, "after meals stand; or walk a thousand feet,"[2] which the Germans have translated better into "Nach der Malzeit zu stehen oder 1000 Schritt zu gehen." Virdiet[3] states "whosoever indulges in violent exercise after eating is apt to feel badly." The first investigations concerning the effect of rest and exercise upon the digestion were made by Beaumont in his famous experiments on St. Martin; on page 87 in the second edition of his work (1847) we read:

On the subject of exercise or repose, during the digestion of a meal, there has been some diversity of opinion. It has generally been conceded, however, that a state of repose is most favorable to chymification. It has been said, that during the digestion of aliment the energies of the system were centered on the stomach, and should not be withdrawn to any

[1] Read at the Annual Meeting of the American Gastroenterological Association, Boston, June 4, 1906.

distant part; that the stomach becomes a center of fluxion, etc. I protest, again, against the use of terms which have no definite meaning. I believe the benefit of science will be better subserved by adhering to facts, and the deductions of experiments, than by the propagation of hypotheses founded on uncertain data. From numerous trials, I am persuaded that moderate exercise conduces considerably to healthy and rapid digestion. The discovery was the result of accident, and contrary to preconceived opinions, I account for it in the following way: Gentle exercise increases the circulation of the system, and the temperature of the stomach. This increase of temperature is generally about 1.5°. Now, if the gastric juice be a solvent, its action is similar to other chemical solvents, and its activity is increased in proportion to the elevation of its temperature. Of the reason, I leave others to judge. The effect is certain. Severe and fatiguing exercise, on the contrary, retards digestion. Two reasons present themselves for this—the debility which follows hard labor, of which the stomach partakes; and the depressed temperature of the system, consequent upon perspiration, and evaporation from the surface.

In 1849, Louis Villain[4] attempted to demonstrate by experiments on animals, that the food is much more thoroughly and quickly digested in the stomach of animals during rest than after exercise. His experiments consisted in giving two dogs at the same time the identical quantity and character of food; while the one animal was forced to run about, the other was allowed to rest quietly. After a certain length of time both animals were killed; the food was found almost in the same condition in the stomach of the animal which had been exercising as it had been before being swallowed,

while it was thoroughly digested and had already formed into chyme in the stomach of the resting animal.

Fleischer[5] showed that it makes but little difference whether one is resting or up and about after eating, the food being equally well digested in either case; he found on the other hand that rapid walking retards digestion. Forster,[6] however, did not note any difference in the digestion of food in the stomach during exercise or rest. Cohn[7] utilized dogs in his investigations. These animals were allowed to rest after meals or were subjected to moderate exercise; a walk of two hours. The gastric contents was removed by lavage with the stomach-tube. He concluded that moderate exercise immediately after meals disturbs gastric digestion. Streng[8] also experimented on dogs to determine more fully the effect of rest and exercise upon digestion. He gave as a test-meal 25 grams of carne pura in 300 cc. of warm water; some animals were kept at complete rest, while others were forced to exercise vigorously for three hours. The stomach was emptied of its contents in three hours. His experiments showed no difference either in the motor function or in the secretion of acid in either class of cases. Streng also experimented with three healthy individuals. Test-meals of 200 grams of scraped beef, a slice of bread, a plate of bouillon, three spoonfuls of mashed potatoes were given and the contents of the stomach was removed in three hours. Those experimented upon were required either to sit quietly or to move about. His results showed no special difference in any case; so

that according to Streng's views the gastric digestion is neither affected by rest nor by exercise.

Salvioli[9] went more elaborately into this question, experimenting on dogs with gastric fistulas. He observed that exercise materially diminishes the quantity of gastric juice secreted; for instance in a resting dog 20 cc. to 30 cc. of gastric juice was obtained in 15 minutes; while in a dog fatigued by exercise only a few drops or at most 5–10 cc. were obtained in the same period of time. He also found that exercise markedly diminished the digestive power of the gastric secretion; for instance, if an equal quantity of egg albumen be placed into the stomach of two dogs through a fistula, and the one dog be allowed to rest and the other to exercise, the albumen in the stomach of the resting dog was digested in three hours, while in the animal which had been exercising large quantities of undigested albumen were found remaining. The difference in digestive power is shown even more clearly when digesting albumen artificially in an incubator at 37° C. The quantity of egg albumen which is digested in six hours by a normal gastric juice, that is from an animal at rest, is not digested in more than ten hours in the gastric secretion from a dog which has been exercising. Moderate exercise does not diminish the gastric secretion, while excessive exercise markedly retards this process. Salvioli also observed that the propulsive force of the stomach was more greatly accelerated in these animals undergoing violent exercise than in those at rest.

Spirig[10] found after performing a number of experiments upon himself and another normal individual that the acidity of the gastric secretion is greater when resting than after performing physical exercise; while the propulsive force of the stomach is accelerated by physical exercise. He therefore advises patients suffering with hyperacidity to exercise moderately after meals.

Schüle[11] investigated more particularly the effect of sleep upon the digestive functions. He gave his patients, who had normal digestive powers, 150 grams of beefsteak, 100 grams of potato purée and 250 cc. of white wine; the stomach was emptied of its contents in four hours. Under normal conditions his patients showed a total gastric acidity of 66 to 90; free HCl, 0.15 to 0.24; after sleeping, the total acidity would range between 120 and 130, while the free hydrochloric acid would rise from 0.29 to 0.31, showing that sleep has a tendency to induce a superacidity. This condition was not produced merely by resting in bed, for the same patients would not show a superacidity under these conditions. Schüle believes that the superacidity, which is observed after sleeping is induced by a lessened gastric motility, the food contents remaining in the stomach stimulating the flow of acid; for he also found that those patients in whom the stomach would ordinarily empty itself entirely of its food contents in six hours, would still contain considerable remains of food in the stomach if required to sleep the same length of time. From his observations, Schüle considers it best for patients suffering with hyperchlorhydria not to sleep after meals,

while those suffering with dilation should rest after eating.

The results obtained by the various observers concerning this subject have been so conflicting, that it occurred to me that this question required further investigation. For this reason certain individuals, two with normal digestive powers and eight suffering with various gastric disturbances, were subjected to the following experiments. They were given a test-dinner consisting of 40 cc. of soup, 200 gm. of beefsteak, 50 gm. of wheat bread and 200 cc. of water; the tests were removed after four hours. The stomach was as completely emptied as possible and then washed with a definite quantity of water and in this way the total amount of contents remaining in the stomach four hours after a test-dinner could easily be ascertained, from which a fairly clear conception of the motor activity could be obtained. The gastric content was then examined for the total acidity and the percentage of free acid. The individuals were examined after resting, after moderate exercise, that is after walking about leisurely, after excessive exercise during a greater part of the time, and finally after sleeping. In order to ensure sound sleep 5 gr. of trional was added to the test-meal in the special experiments; only, however, after ascertaining that this drug had no appreciable effect either on the motor or secretory function of the stomach.

Normal Cases.—There were two normal individuals subjected to our experiments.

NORMAL CASE I.—Patient, H.F.

Experiment No. 1: Rest after meals. Quan-

tity of contents removed, 50 cc. Total acidity, 55. Percentage of free HCl, 0.111.

Experiment No. 2: Rest after meals. Quantity of contents removed, 44 cc. Total acidity, 58. Percentage of free HCl, 0.120.

Experiment No. 3: Moderate exercise after meals. Quantity of contents removed, 42 cc. Total acidity, 58. Percentage of free HCl, 0.123.

Experiment No. 4: Moderate exercise after meals. Quantity of contents removed, 48 cc. Total acidity, 54. Percentage of free HCl, 0.112.

Experiment No. 5: Excessive exercise after meals. Quantity of contents removed, 88 cc. Total acidity, 50. Percentage of free HCl, 0.111.

Experiment No. 6: Excessive exercise after meals. Quantity of contents removed, 74 cc. Total acidity, 54. Percentage of free HCl, 0.115.

Experiment No. 7: Sleep after meals. Quantity of contents removed, 80 cc. Total acidity, 52. Percentage of free HCl, 0.118.

Experiment No. 8: Sleep after meals. Quantity of contents removed, 84 cc. Total acidity, 55. Percentage of free HCl, 0.116.

NORMAL CASE II.—Patient, H.A.

Experiment No. 1: Rest after meals. Quantity of contents removed, 70. Total acidity, 40. Percentage of free HCl, 0.121.

Experiment No. 2: Rest after meals. Quantity of contents removed, 62 cc. Total acidity, 44. Percentage of free HCl, 0.110.

Experiment No. 3: Moderate exercise after meals. Quantity of contents removed, 74 cc. Total acidity, 52. Percentage of free HCl, 0.120.

Experiment No. 4: Moderate exercise after meals. Quantity of contents removed, 60 cc. Total acidity, 50. Percentage of free HCl, 0.119.

Experiment No. 5: Excessive exercise after meals. Quantity of contents removed, 84 cc. Total acidity, 48. Percentage of free HCl, 0.117.

Experiment No. 6: Excessive exercise after meals. Quantity of contents removed, 90 cc. Total acidity, 46. Percentage of free HCl, 0.123.

Experiment No. 7: Sleep after meals. Quantity of contents removed, 100 cc. Total acidity, 52. Percentage of free HCl, 0.110.

Experiment No. 8: Sleep after meals. Quantity of contents removed, 98 cc. Total acidity, 50. Percentage of free HCl, 0.113.

The results as shown in Cases I and II are practically identical. During rest and moderate exercise neither the secretory nor the motor function of the stomach is in any way affected. The amount of contents removed from the stomach varies between 50 cc. and 74 cc., the total acidity between 40 and 55, the percentage of free acid is about 0.1. After excessive exercise or sleep, the motor function of the stomach was found somewhat impaired, as much as from 68 cc. to 100 cc. of contents having been obtained; the secretory function remained normal; the total acidity ranging between 46 and 55 and the percentage of free acid being about 0.1.

It is evident, therefore, that under normal conditions the secretory as well as the motor functions of the stomach are not interfered with during rest; after violent exercise or during sleep after a meal, however, the secretory function remains normal, but the motor function is slightly disturbed.

Gastric Disturbance.—Two patients suffering

with hyperchlorhydria with normal motor function were examined. The results are shown in Cases III and IV.

CASE III.—*Hyperchlorhydria with Normal Motility.*—Patient, J. M.

Experiment No. 1: Rest after meals. Quantity of contents removed, 60 cc. Total acidity, 70. Percentage of free HCl, 0.123.

Experiment No. 2: Rest after meals. Quantity of contents removed, 74 cc. Total acidity 74. Percentage of free HCl, 0.134.

Experiment No. 3: Moderate exercise after meals. Quantity of contents removed, 80 cc. Total acidity, 82. Percentage of free HCl, 0.281.

Experiment No. 4: Moderate exercise after meals. Quantity of contents removed, 86 cc. Total acidity, 83. Percentage of free HCl, 0.264.

Experiment No. 5: Excessive exercise after meals. Quantity of contents removed, 92 cc. Total acidity, 82. Percentage of free HCl, 0.256.

Experiment No. 6: Excessive exercise after meals. Quantity of contents removed, 94 cc. Total acidity, 88. Percentage of free HCl, 0.279.

Experiment No. 7: Sleep after meals. Quantity of contents removed, 100 cc. Total acidity, 84. Percentage of free HCl, 0.266.

Experiment No. 8: Sleep after meals. Quantity of contents removed, 96 cc. Total acidity, 88. Percentage of free HCl, 0.287.

CASE IV.—*Hyperchlorhydria with Normal Motility.*—Patient, S.L.

Experiment No. 1: Rest after meals. Quantity of contents removed, 56 cc. Total acidity, 71. Percentage of free HCl, 0.110.

Experiment No. 2: Rest after meals. Quantity of contents removed, 60 cc. Total acidity, 68. Percentage of free HCl, 0.135.

Experiment No. 3: Moderate exercise after meals. Quantity of contents removed, 72 cc. Total acidity, 94. Percentage of free HCl, 0.291.

Experiment No. 4: Moderate exercise after meals. Quantity of contents removed, 68 cc. Total acidity, 92. Percentage of free HCl, 0.289.

Experiment No. 5: Excessive exercise after meals. Quantity of contents removed, 95 cc. Total acidity, 88. Percentage of free HCl, 0.288.

Experiment No. 6: Excessive exercise after meals. Quantity of contents removed, 100 cc. Total acidity, 90. Percentage of free HCl, 0.293.

Experiment No. 7: Sleep after meals. Quantity of contents removed, 115 cc. Total acidity, 82. Percentage of free HCl, 0.272.

Experiment No. 8: Sleep after meals. Quantity of contents removed, 104 cc. Total acidity, 85. Percentage of free HCl, 0.261.

While resting the motor function remains normal, from 56 cc. to 74 cc. of contents being obtained; while the total acidity was reduced, ranging between 68 and 74; the free hydrochloric acid being about 0.13 percent. During moderate exercise the motor function remains about normal; 72 cc. to 86 cc. being obtained, while the total acidity remains about the same (82 to 94), the percentage of free hydrochloric acid being from 0.26 to 0.29. During excessive exercise as well as sleep, the motor function is markedly impaired, 92 cc. to 115 cc. being obtained, while the secretory function remains unchanged, the total acidity ranging from 82 to 88, free hydrochloric acid being about 0.25 to 0.29 percent.

It is therefore shown that in cases of hyperchlorhydria with normal motor function, the acidity is reduced, the motor function remaining normal while resting; the acidity remains unaltered, the motor function being normal during moderate exercise; while the acidity is about the same and the motor function is markedly impaired during excessive exercise and during sleep.

In cases of hyperchlorhydria with motor insufficiency the following observations were made upon two patients.

CASE V.—*Hyperchlorhydria with Motor Insufficiency.*—Patient, J.S.

Experiment No. 1: Rest after meals. Quantity of contents removed, 110 cc. Total acidity, 72. Percentage of free HCl, 0.213.

Experiment No. 2: Rest after meals. Quantity of contents removed, 115 cc. Total acidity, 74. Percentage of free HCl, 0.224.

Experiment No. 3: Moderate exercise after meals. Quantity of contents removed, 100 cc. Total acidity, 84. Percentage of free HCl, 0.264.

Experiment No. 4: Moderate exercise after meals. Quantity of contents removed, 97 cc. Total acidity, 82. Percentage of free HCl, 0.253.

Experiment No. 5: Excessive exercise after meals. Quantity of contents removed, 130 cc. Total acidity, 85. Percentage of free HCl, 0.295.

Experiment No. 6: Excessive exercise after meals. Quantity of contents removed, 128 cc. Total acidity, 81. Percentage of free HCl, 0.253.

Experiment No. 7: Sleep after meals. Quantity of contents removed, 125 cc. Total acidity, 89. Percentage of free HCl, 0.274.

Experiment No. 8: Sleep after meals. Quantity of contents removed, 118 cc. Total acidity, 94. Percentage of free HCl, 0.283.

CASE VI.—*Hyperchlorhydria with Motor Insufficiency.*—Patient, J.J.

Experiment No. 1: Rest after meals. Quantity of contents removed, 100 cc. Total acidity, 66. Percentage of free HCl, 0.204.

Experiment No. 2: Rest after meals. Quantity of contents removed, 98 cc. Total acidity, 62. Percentage of free HCl, 0.210.

Experiment No. 3: Moderate exercise after meals. Quantity of contents removed, 88 cc. Total acidity, 78. Percentage of free HCl, 0.234.

Experiment No. 4: Moderate exercise after meals. Quantity of contents removed, 90 cc. Total acidity, 80. Percentage of free HCl, 0. 257.

Experiment No. 5: Excessive exercise after meals. Quantity of contents removed, 116 cc. Total acidity, 82. Percentage of free HCl, 0.250.

Experiment No. 6: Excessive exercise after meals. Quantity of contents removed, 118 cc. Total acidity, 88. Percentage of free HCl, 0.261.

Experiment No. 7: Sleep after meals. Quantity of contents removed, 110 cc. Total acidity, 98. Percentage of free HCl, 0.285.

Experiment No. 8: Sleep after meals. Quantity of contents removed, 114. Total acidity, 96. Percentage of free HCl, 0.289.

While resting, the motor function was found impaired, 98 cc. to 115 cc. of contents being obtained, while the total acidity was diminished, varying between 62 and 74, the percentage of free hydrochloric acid being 0.20 to 0.22; during moderate exercise the motor function remains unaltered, 88 cc. to 100 cc. of contents being

obtained, while the acidity also remains unchanged, 78 to 84, the percentage of free hydrochloric acid being 0.23 to 0.26. During excessive exercise, the motor function is still further impaired, 116 cc. to 130 cc. of contents being obtained, while the acidity remains about the same, the total acidity varying between 81 and 88; free hydrochloric acid 0.25 percent. During sleep the motor function is also greatly impaired, 114 cc. to 125 cc. of contents being found; while the total acidity is further increased from 89 to 94; free hydrochloric acid, 0.27 percent. to 0.28 percent.

In conditions of hyperchlorhydria with motor insufficiency the acidity is lessened and the motor function impaired during rest; the acidity and motor function remain unaltered during moderate exercise; the acidity remains unchanged while the motor function is still further impaired during excessive exercise; the acidity is still further increased and the motor function is still further impaired during sleep.

The effect of rest, exercise, and sleep was also studied in two cases of gastric catarrh with normal function. The results are shown in Cases VII and VIII:

CASE VII.—*Catarrh with Normal Motility.*—Patient, S.J.

Experiment No. 1: Rest after meals. Quantity of contents removed, 64 cc. Total acidity, 24.

Experiment No. 2: Rest after meals. Quantity of contents removed, 62 cc. Total acidity, 28.

Experiment No. 3: Moderate exercise after meals. Quantity of contents removed, 54 cc. Total acidity, 10.

Experiment No. 4: Moderate exercise after meals. Quantity of contents removed, 59 cc. Total acidity, 12.

Experiment No. 5: Excessive exercise after meals. Quantity of contents removed, 92 cc. Total acidity, 11.

Experiment No. 6: Excessive exericise after meals. Quantity of contents removed, 96 cc. Total acidity, 9.

Experiment No. 7: Sleep after meals. Quantity of contents removed, 105 cc. Total acidity, 34.

Experiment No. 8: Sleep after meals. Quantity of contents removed, 115 cc. Total acidity, 36.

CASE VIII.—*Catarrh with Normal Motility.*—Patient, H.H.

Experiment No. 1: Rest after meals. Quantity of contents removed, 55 cc. Total acidity, 36.

Experiment No. 2: Rest after meals. Quantity of contents removed, 58 cc. Total acidity, 38.

Experiment No. 3: Moderate exercise after meals. Quantity of contents removed, 62 cc. Total acidity, 20.

Experiment No. 4: Moderate exercise after meals. Quantity of contents removed, 50 cc. Total acidity, 24.

Experiment No. 5: Excessive exercise after meals. Quantity of contents removed, 99 cc. Total acidity, 21.

Experiment No. 6: Excessive exercise after meals. Quantity of contents removed, 104 cc. Total acidity, 24.

Experiment No. 7: Sleep after meals. Quantity of contents removed, 88 cc. Total acidity, 38.

Experiment No. 8: Sleep after meals. Quantity of contents removed, 96 cc. Total acidity, 42.

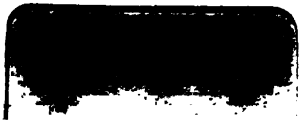
While resting, the motor function was found to remain normal, from 55 to 64 cc. of contents being obtained; the acidity being increased, the total acidity ranging between 28 and 38; during moderate exercise the motor function remained normal, from 50 cc. to 60 cc. of contents being obtained, the acidity also remained about the same as it was originally, from 10 to 24. During excessive exercise the motor function is markedly impaired, from 92 cc. to 104 cc. of contents being obtained, while the total acidity remains about the same, from 9 to 24. During sleep, the motor function is also markedly impaired, the quantity of contents obtained ranging from 88 to 115, while the total acidity is still further increased 34 to 42.

It is therefore evident that in cases of hypochlorhydria with normal motor activity, the acidity is increased and the motor function remains normal during rest; the acidity remains unaltered while the motor function remains normal during moderate exercise; the acidity remains unchanged while the motor function is markedly impaired during excessive exercise; the acidity is still further increased and the motor function markedly impaired during sleep.

The results obtained from two patients suffering with hypochlorhydria with disturbed motor function, upon whom the effect of rest, exercise and sleep was studied are represented in Cases IX and X.

CASE IX.—*Catarrh with Motor Insufficiency.*—Patient, V.R.

Experiment No. 1: Rest after meals. Quantity of contents removed, 81 cc. Total acidity, 38.



Experiment No. 2: Rest after meals. Quantity of contents removed, 83 cc. Total acidity, 36.

Experiment No. 3: Moderate exercise after meals. Quantity of contents removed, 98 cc. Total acidity, 16.

Experiment No. 4: Moderate exercise after meals. Quantity of contents removed, 105 cc. Total acidity, 20.

Experiment No. 5: Excessive exercise after meals. Quantity of contents removed, 144 cc. Total acidity, 18.

Experiment No. 6: Excessive exercise after meals. Quantity of contents removed, 122 cc. Total acidity, 22.

Experiment No. 7: Sleep after meals. Quantity of contents removed, 135 cc. Total acidity, 15.

Experiment No. 8: Sleep after meals. Quantity of contents removed, 150 cc. Total acidity, 19.

CASE X.—*Catarrh with Motor Insufficiency.*—Patient, W.L.

Experiment No. 1: Rest after meals. Quantity of contents removed, 68 cc. Total acidity, 22.

Experiment No. 2: Rest after meals. Quantity of contents removed, 72 cc. Total acidity, 30.

Experiment No. 3: Moderate exercise after meals. Quantity of contents removed, 110 cc. Total acidity, 12.

Experiment No. 4: Moderate exercise after meals. Quantity of contents removed, 114 cc. Total acidity, 14.

Experiment No. 5: Excessive exercise after meals. Quantity of contents removed, 158 cc. Total acidity, 13.

Experiment No. 6: Excessive exercise after meals. Quantity of contents removed, 134 cc. Total acidity, 14.

Experiment No. 7: Sleep after meals. Quantity of contents removed, 152 cc. Total acidity, 11.

Experiment No. 8: Sleep after meals. Quantity of contents removed, 135 cc. Total acidity, 12.

While resting the motor function is impaired, 68 cc. to 83 cc. of contents being obtained; the acidity is increased, the total acidity ranging from 22 to 36; during moderate exercise the motor function remains unchanged, from 98 cc. to 114 cc. of contents being obtained; the total acidity also remains about the same, 12 to 20; during excessive exercise the motor function is markedly impaired, from 122 cc. to 158 cc. of contents being obtained, the total acidity ranging between 13 and 22; during sleep the motor function is still further impaired, 135 cc. to 152 cc. of contents being obtained, while the total acidity remains unchanged, 11 to 19. It is evident, therefore, that in cases of hypochlorhydria with disturbed motor function the acidity seems to be increased and the motor function impaired during rest, the acidity and the motor function remaining unchanged during moderate exercise, the acidity remaining unchanged while the motor function is markedly impaired during excessive exercise, the acidity remaining unchanged while the motor function is further impaired during sleep.

From these observations, the following conclusions may be drawn:

1. In persons with normal digestive powers it makes but little difference whether the individuals rest, exercise, or sleep after meals;

though after violent exercise or sleep the gastric digestion is very slightly impaired.

2. In patients suffering with superacidity and subacidity it is best to order rest after meals; after violent exercise or during sleep, the digestion is impaired in these cases.

3. In patients suffering with motor disturbances of the stomach, it is best to prescribe moderate exercise after meals, for rest, violent exercise, or sleep disturbs the digestion under these conditions.

LITERATURE.

[1] "Coctioni magis conducere quietem."

[2] "Post caenam stabis vel mille passus deambulabis."

[3] "Eadem causa male digerunt, qui post pastum motibus violentis indulgent." (Johannis Virideti. Tractabus novus medico physicus de prima coctione, praecipueque deventriculo fermento.) Genevae, mdcxci, page 312.

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Reprinted from MEDICAL RECORD, September 1, 1906.

HABITUAL CONSTIPATION, VIEWED FROM THE STANDPOINT OF MODERN EVOLUTION OF DIETETICS, IS A PHYSIOLOGICAL PHENOMENON.*

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THE conception of regular and irregular movements of the bowels is vague and indefinite as far as the physician is concerned. A movement of the bowels once a week, or even at longer intervals, has been called regular by many of our patients, and one or two movements daily are often designated as constipation, if these movements leave the patient in an unsatisfied and ever-longing state of mind. We have no clinical methods by means of which the physician could make a diagnosis of constipation, and every intestine, therefore, is a law unto itself. The physician, when assuming the responsibility of treating a case of constipation, is guided by the statement of his patient; he accepts the definition of the word as applied by the sufferer to his particular case. Indeed, the physician has no choice in the matter and must accept the statement of the patient; there is not another disease in the whole range of medicine where malingering could be practised with as little

* Read at the ninth annual meeting of the American Gastroenterological Association, held at Boston, June 4 and 5, 1906.

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danger of detection. One who practises rational medicine will endeavor to find out the etiological factors which may give rise to this condition, and, if found, treat the constipation by removing the cause. If, however, after a thorough examination of the patient, no pathological condition is found which would justify the existence of constipation as a symptom, we simply prefix the term "constipation" by the word "habitual," and thereby raise it to the dignity of a disease *sui generis*. Having cleared our conscience from the responsibility of looking for a cause, we start out with our energetic treatment. We change the mixed diet to one containing a large percentage of residue of indigestible substances; we increase the quantity and frequency of meals; we use ether spray over the abdomen; we stretch the sphincter, we insufflate, inflate, dilate, and plug the rectum; in short, we exercise our ingenuity to irritate the bowel, dietetically, thermically, mechanically, electrically, etc., so as to increase its peristaltic movements, and induce premature delivery of the fecal matter. Here, too, we encounter another difficulty. Since the number of evacuations within any given time does not constitute a criterion of normal or abnormal, the question which each physician has to solve is, How often must the bowels move to justify the statement that the patient is cured? All physicians endeavor to secure one movement every day, and anything less than that is not considered a successful cure. Why should one movement a day be considered normal? Why not two movements in one day, or one movement in two days? By what are we to be guided? As in the case of diagnosis, the physician must be guided by the patient; so it is with the treatment. The physician is at the mercy of the patient. He it is who is to decide when he is cured or not; and upon his word depends the suc-

cess or failure of the treatment. Is it not possible that the many failures to cure constipation which every physician has to his discredit, are due to the fact that these patients would do well with a movement of the bowels once in two or three days?

Eminent clinicians and investigators like Leichtenstern, Ewald, Dunin, Meinert, Glenard, and others have each brought forward an ingenious theory as to the causation of habitual constipation, all of which remain mere conjectures. Nothnagel makes the broad statement that habitual constipation is "a primary and independent form of disease, and must be considered as such." But this problem will not be solved until we have learned more about feces. It is a sign of the times that within the last two years have appeared two editions of Schmidt's and Strassburger's work on feces and more than a dozen theses and monographs on the subject.

At a recent meeting of the Naturforscher Gesellschaft Ad. Schmidt declared that all the theories as to the causation of constipation hitherto proposed are untenable, and that the only rational explanation of this obscure condition is to be found in the theory of intensive utilization (*intensive ausnutzung*) by the bowel of the nutritive ingredients contained in the food, which explains the small quantity and dry quality of the fecal matter found in the majority of cases of true habitual constipation.

Now, the question arises, is intensive utilization a pathological or physiological phenomenon? For some time I have been conducting a series of observations on patients suffering from habitual constipation, with a view of ascertaining the quantity of food ingested and the corresponding quantity of fecal matter expelled.

The factors that enter into these studies are so many and varied that it will require many more ob-

servations to be recorded and a very careful sifting of the facts and figures, before they can be tabulated and made available. The greatest difficulty which one meets from the outset is the absence of a standard as to either quantity or quality of fecal matter in the healthy individual, so that there is nothing to compare with or be guided by. Then, each ingredient of food produces a certain quantity and quality of fecal matter. This is an important study which has been attempted only lately by Lynch, and which will yield valuable results in the future. Lastly, each healthy man possesses a certain capacity for the amount of food ingested and absorbed, as well as for fecal matter produced, the relation between the two not being a constant number. Many persons eat large meals and produce scant movements of the bowels, and vice versa.

The observation on the rectopath is made more difficult because he or she whose mind and soul dwell in the nether regions does not "take much stock" in the scientific side of the question, and therefore, with the exception of a few intelligent persons, does not permit one to observe the course of constipation under normal conditions. They insist on immediate change of diet, the use of laxatives, enemas, all of which create an abnormally abnormal condition.

But, notwithstanding the fact that I am not ready to present a finished report of my labors, I have already gathered enough data to form in my mind a composite picture of the sufferers from habitual constipation and to assert that in patients thus afflicted, especially males who work hard, enjoy a good appetite, sleep well, and who keep up their bodily weight, the constipation is due to physiological causes, *i.e.* the food they ingest is thoroughly utilized, little residue is left, and it is natural for such patients to have a movement of the bowels at

intervals of two, three, or more days. Such a condition cannot be regarded as pathological. To prescribe drugs, a laxative diet, or the various other irritants means eventually to induce a catarrhal condition of the bowels.

The question naturally arises, if the small quantity of fecal matter and the long intervals between the movements is a physiological condition, it should not give rise to any inconvenience or discomfort and we would not expect such a normal man to seek the services of a physician.

I will explain this by a hypothetical case: A man who is accustomed to have a movement of the bowels, say once a day immediately after breakfast, is called away immediately after breakfast, or must go without breakfast. What happens? The nervous mechanism of the intestine was wound up by the clock-work habit for twenty-four hours. Something interfered. The bowel is irritable for an hour or longer, the man feel uncomfortable, and gradually the irritation subsides, and the discomfort it occasions disappears. If the subject does not pay any attention to it—if he does not consider himself in duty bound to have a movement of the bowel once a day and every day of his life, he will go to sleep and wait for the next morning until after breakfast, when a regular movement, probably larger in quantity will be the result. If, however, instead of letting nature take care of itself, the man will come home in the evening and begin to make efforts and strain to have a bowel movement, or will take a laxative, insert a suppository, etc., he will at once create an abnormal condition. If he induces artificially a movement of the bowels, say late at night, he will in all probability miss his regular movement the following morning, notwithstanding the extra straining he may do. Again he

may induce a movement in the evening and thus start up constipation. In this case the little discomfort which the patient experienced at the beginning and which every healthy body is and should be able to throw off without leaving any trace behind, has started on its way a case of habitual constipation; the patient interpreted the temporary discomfort he experienced as the outset of a grave malady, "constipation," to ward against which he immediately instituted energetic prophylactic measures.

This goes to illustrate that we can cultivate two kinds of habits, and especially in relation to movements of the bowels. One habit is that the bowels should move at regular intervals, and the other is the habit of breaking that habit whenever necessary. As Sir James Paget (Studies of Old Case Books) said: "These half involuntary organs (rectum and bladder) are good servants but very bad masters; the more they are indulged the more peremptory they become; . . . they should be educated to subserviency; . . . any one with a resolute will may easily teach them."

This sort of constipation is simply due to a loss of faith in one's self and in the elasticity and pliancy in the functions of the body, to a fear that a slight disturbance from the usual habits may lead to profound pathological changes. It is a fear of constipation—a phobia.

There are others who think they suffer from constipation simply because they have made up their minds that they will not be cured unless they have one movement daily.

Humanity is undergoing profound changes in regard to dietetics. We may consider the last century as a transition period. At no time in the past have the civilized nations consumed so much meat, eggs, milk, and especially sugar as during the last century,

and the use of the latter article is growing from year to year. The number of predigested, prepared, dextrinized foods is increasing daily. The high efficiency of the dental art and the greater stress laid upon the proper mastication of food all these factors produce certain changes in the human organism, which manifest themselves by a reduced quantity of the fecal matter and less frequent need for emptying the bowels.

From the above consideration, I wish to make the following deductions:

Habitual constipation in a good many cases is due to a mistaken diagnosis. The patient makes the diagnosis himself based upon his sensation only. Once the diagnosis of constipation is made, and then corroborated by the physician, the diagnosis becomes an etiological factor.

The tendency of civilized life in general and of modern dietetics in particular is toward the production of lesser quantities of fecal matter and less frequent intervals of evacuation.

There is no doubt that the habit of regularity is beneficent, but no matter what one's philosophy is regarding free will, it is right that men should resolve that they will not allow every unusual sensation to become a source of morbid concentration.

The treatment of habitual constipation should be directed more toward rectifying the errors of the mind than those of the body; the fear engendered in the patient of an impending calamity should be allayed by impressing upon him the simple, physiological truth that certain foods are better absorbed than others, that it is not imperative for the preservation of good health to have a movement of the bowels daily, nor is the small quantity indicative of disease.

DISCUSSION.

DR. A. D. KOHN, OF CHICAGO: I would take issue with the reader of the paper on one point, and that is in regard to the question of sugar. I think an increased amount of sugar would rather tend to open the bowels than to constipate them: at least, that has been my experience.

DR. J. DUTTON STEELE, OF PHILADELPHIA: What I have to say in connection with Dr. Spivak's interesting paper is not quite germane to the subject. What I have in mind is the difficulty in determining the functional derangements of the intestine by the methods generally in vogue. In my experience with the Schmidt diet, I have found it open to the criticism that it is very constipating, and I do not agree with him that the excess of milk causes diarrhoea. I have never seen that. In fact I have had difficulty to produce an evacuation of the bowels after such a meal, and have even failed to get the meal through in time by using a glycerine suppository, and frequently I have had to fall back on rhubarb and soda or Carlsbad salts.

Schmidt, in speaking of the stools in jaundice, makes the statement that they are non-irritating because of the excess of fat and soap they contain. This he refers to as a contradistinction to the character of the stools in pancreatic disease. In three or four cases of chronic jaundice that were under my observation, the patients were inclined to be constipated, and the stools contained an excess of fatty acids and some soap.

I have been very much impressed by the futility of trying to recognize pancreatic disease by studying the fatty contents of the stools. I recall one interesting case of carcinoma of the pancreas, with the formation of a biliary fistula, in which there was a complete absence of bile from the intestines. An operation on the gall-bladder was done, and as soon as bile began to flow into the intestine, the neutral fats entirely disappeared, which shows conclusively that the bile has an extremely important effect on the fats in the intestine.

DR. MAX EINHORN, OF NEW YORK CITY: I want to say a few words in regard to the Schmidt theory of habitual constipation, according to whose idea, practically, the constipation is due to the fact that too much of the food is utilized, and that there is consequently too little residue. I think that is rather a result of constipation than a cause. If the food products remain in the bowel too long, the fluids and other elements are absorbed. Many people are constipated because they take too little vegetable material.

Dr. Spivak referred to good teeth, slow eating, etc., as causes of constipation. I do not thnik they can be regarded as real etiological factors, because it was no doubt originally intended that the human being should have good teeth. The reason why so many people have

to depend on false teeth is that they were brought up on a faulty diet, and did not make enough use of their natural teeth.

I do not see any objection to the use of the term habitual constipation. Dr. Spivak himself says that in order to treat habitual constipation, we must first make the diagnosis. In habitual constipation, as I understand it, there is no organic lesion, and the condition is induced by some fault in the diet or in the the manner of living, and in order to overcome it, we must educate the patient and put him on the proper diet. At the same time he should be told not to worry, even if he does not have a movement of the bowels every day or two.

Dr. S. J. Meltzer: In connection with this subject I might mention the case of a man whose bowels move sometimes only about once every four weeks; this has continued for years, and he has apparently suffered no ill-effects therefrom.

Dr. G. W. McCaskey, of Fort Wayne, Ind.: The paper of Dr Spivak was very interesting and suggestive, but I do not think I can quite follow him to his ultimate conclusions. It seems to me in the first place that there is a good physiological reason for expecting an emptying of the bowels once a day, although I do not wish to imply that a failure to do this will be followed by serious consequences. We may meet with remarkable instances of constipation, like the one mentioned by Dr. Meltzer, where the failure to empty the bowels is apparently not followed by any untoward symptoms, but the average individual takes his food three times a day—morning, noon and night—and under strictly physiological conditions that food will serve its purpose, and its indigestible residue will be ready for expulsion in about twenty-four hours. That is the average condition of the human race, I think under civilized conditions, and any wide departures from it are exceptional and objectionable. There are many reasons for this. The too long transit of the food through the intestines gives more opportunity for the production of toxins, which are undoubtedly, in my opinion, the cause of many chronic ailments to which these patients are subject. After the food reaches the transverse colon, where, according to Dr. Cannon's researches, the residue becomes hard, and especially when it gets a little further down, then no great harm can come from it, so far as auto-intoxication is concerned, but above that region, in the transverse colon and higher up, the danger of auto-intoxication is apparent, and may give rise to serious types of chronic disease. I do not, therefore, think it a safe postulate to assume that it is physiological to have a movement of the bowels every two, three, four or five days. Exceptional instances may be recorded where such a condition of affairs may exist for years without any apparently serious results, but the time may come when the individual's resistive power is lessened to such a degree that he will be apt to suffer.

The diet of our day is undoubtedly conducive to constipation. I do not think the so-called prepared foods, meaning thereby the various cereals on the market, are especially conducive to constipation, because they contain, substantially, the same amount of indigestible residue as other foods, unless they are first submitted to a high temperature. The predominance of meat and eggs, however, in our modern dietary are objectionable features. I myself suffer from gout, and at times meet with considerable difficulty in finding enough of suitable food to eat. While on a recent visit to Spain, the menus that I had to select from were made up largely of meat dishes, and as the result of my forced indiscretions in diet, I suffered seriously from constipation and gout. Our usual diet should contain more indigestible debris than it does. Such residue is largely found in vegetables and the better class of cereals.

I believe we should have a regular evacuation of the bowels each day. The size of the evacuation,—the quantity of the alvine discharge—will of course depend greatly on the amount of indigestible debris. There is no necessity for a large movement, but there should be a movement. There should be an expulsion about once in the course of twenty-four hours. That, I think, is the ideal condition.

Dr. John A. Lichty, of Pittsburg: I think Dr. Spivak has established his point that the modern evolution of dietetics has something to do with constipation, which seems to be on the increase. Whether or not we should call constipation the condition where the bowels have not moved in two or three days is a question that depends on the feelings of the patient. Many who have become accustomed to such a condition would doubtless feel uncomfortable if you disturbed them in their habits, and I think it is better to leave them alone.

Our modern method of living, I think, has much to do with the frequency with which constipation occurs, especially among certain classes of people. Shop-girls, stenographers, and others who must be at their place of business promptly at a certain hour usually allow themselves just enough time to rush from the breakfast table to the station, and thence to their work, and in his way the calls to stool are too often ignored, and such habits will eventually lead to chronic constipation. Perhaps a remedy will be found for this when we get rid of the suburban trains and secure the benefit of a better trolley service.

I do not know of any more interesting reading than the experiments of Pawlaw on the glands and ferments of the digestive tract. It strikingly shows the delicate arrangement of the glands, especially in the first portion of the bowel. I think it would be a good idea if Pawlaw's deductions were printed and pasted on every pill-box and package that comes from the manufacturing chemist. It would at least impress upon the physician the enormity of his offense when he disturbs the delicate mechanism